



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

Per 1519 @ 113

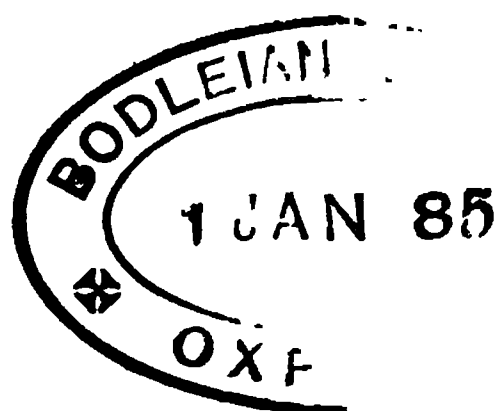
GUY'S HOSPITAL REPORTS.

EDITED BY
FREDERICK TAYLOR, M.D.
AND
N. DAVIES-COLLEY, M.A., M.C.

VOL. XLII,
BEING
VOL. XXVII OF THE THIRD SERIES

LONDON:
J. & A. CHURCHILL, NEW BURLINGTON STREET.

MDCCCLXXXIV.



CONTENTS.

	PAGE
In Memoriam—Charles Hilton Fagge	xxv
I. Surgical Reminiscences. By EDWARD COCK	1
II. Case of probable Thrombosis of Superior Mesenteric Vein and Renal Veins. Detachment of Several Valvulæ Conniventes of Jejunum. Recovery. By Sir WILLIAM W. GULL, Bart., M.D.	15
III. On a Case of Fracture of the Skull, in which Cerebro- spinal Fluid escaped from the Vault fourteen days after Trephining. By R. CLEMENT LUCAS, B.S.	28
IV. Gangrene of the Skin and Cellular Tissue of the Arm following Vaccination. By R. CLEMENT LUCAS, B.S.	31
V. An Account of the Abnormalities observed in the Dissecting Room during the Winter Session 1882-83. By P. HORROCKS, M.D., W. HALE WHITE, M.D., and W. A. LANE, M.S.	39
VI. The Theory of a Heat Centre from a Clinical Point of View. By W. HALE WHITE, M.D.	49
VII. List of Specimens added to the Pathological Museum during the Year 1882-83. By JAMES F. GOOD- HART, M.D.	101
VIII. Cervical and Bicipital Ribs in Man. By ARBUTHNOT LANE, M.S.	109
XI. Remarks upon the Relationship between the Struc- tural Changes and the Clinical Symptoms of Chronic Renal Disease associated with Dropsy. By JAMES F. GOODHART, M.D.	135

	PAGE
X. Notes of Gynæcological Out-patient Cases. By P. HORROCKS, M.D.	157
XI. Hypochondriasis and Hypochondriacal Insanity. By GEORGE H. SAVAGE, M.D.	175
XII. Note of two Cases of General Anasarca in Children without Albuminuria. By JAMES F. GOOD- HART, M.D.	197
XIII. On Albuminuria and the Symptoms which indicate its Gravity. By F. A. MAHOMED, M.B. Cantab.	201
XIV. On Transfusion of Blood for Puerperal Hæmorrhage. By ALFRED LEWIS GALABIN, M.D.	255
XV. Case of Typhoid Fever, fatal on the Seventy-sixth Day, with Hæmorrhage due to recent Typhous Ulceration in the Ileum. By Sir WILLIAM W. GULL, Bart., M.D.	271
XVI. Cases illustrating the Various Results of Partial Sub- cutaneous Laceration of Arteries. By CHARTERS J. SYMONDS, M.S.	275
XVII. On a Case of Hypertrophic Cirrhosis, with Remarks on the Pathology of Cirrhosis. By J. A. P. PRICE, B.A., M.B.	295
XVIII. A Case of Enlarged, so-called "Hypertrophic" Cir- rhotic Liver, with Observations on the Concurrence of Fever with Cirrhosis. By R. E. CARRING- TON, M.D.	337
XIX. A Case of Extensive Cerebral Softening, with Descending Sclerosis in the Lateral Column. By G. N. PITT, M.A., M.D.	351
XX. Observations on the Determination of the Hearing Power. By W. LAIDLAW PURVES, M.D.	357
XXI. A Case of Hydrophobia, in which the Condition of the Larynx was observed during a Spasm. By G. N. PITT, M.A., M.D.	361
XXII. On a Case of Gastrostomy, in which the Artificial Opening was subsequently closed. By N. DAVIES- COLLEY, M.C.	367

Contents.

v

	PAGE
XXIII. On Colles's Fracture. By R. CLEMENT LUCAS, B.S.	375
XXIV. Cases of Aortic Aneurysm opening into the Pulmonary Artery. By FREDERICK TAYLOR, M.D.	391
XXV. On Hernia of the Appendix Vermiformis. By N. DAVIES-COLLEY, M.C.	429
XXVI. List of Specimens added to the Pathological Museum during the Year 1883-84. By JAMES F. GOODHART, M.D.	445
Statistical Summary of Patients treated in Guy's Hospital during the year 1888.	459
List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1882	461
List of Prizemen and Medallists, 1882	467
List of Pupils who have received Appointments at Guy's Hospital, 1882	468
List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1883	478
List of Prizemen and Medallists, 1883	479
List of Pupils who have received Appointments at Guy's Hospital, 1883	480

LIST OF PLATES.

	TO PAGE PAGE
SIR W. W. GULL.	
Plate illustrating his case of Probable Thrombosis of the Superior Mesenteric Vein and Renal Veins .	22
MR. LUCAS.	
Plate illustrating his case of Fracture of the Skull .	30
MR. LUCAS.	
Fig. 1, illustrating his case of Gangrene of the Skin following Vaccination .	} 38
Fig. 2, illustrating an unusual case of Colles's Fracture, page 375 .	
DR. WHITE.	
Plates I and II, Charts of Temperature, illustrating his paper on the Heat Centre .	100
SIR W. W. GULL.	
Plate illustrating his case of Typhoid Fever .	274
DR. PRICE.	
Plate illustrating his paper on Hypertrophic Cirrhosis .	336
MR. LUCAS.	
Plate illustrating his paper on Colles's Fracture .	390

LIST OF WOODCUTS.

	PAGE
DR. HORROCKS, DR. WHITE, and MR. LANE. Five Woodcuts, illustrating their paper on the Abnor- malities observed in the Dissecting-Room	39—48
MR. LANE. Eight Woodcuts, illustrating his paper on Cervical and Bicipital Ribs in Man	107—127
DR. MAHOMED. Thirteen Woodcuts of Sphygmographic Tracings, illus- trating his paper on Albuminuria	202—248
DR. GALABIN. Woodcut, illustrating his paper on Transfusion of Blood	267
MR. SYMONDS. Three Woodcuts, illustrating his paper on Subcutaneous Laceration of Arteries	276—290
DR. F. TAYLOR. Woodcut of Sphygmographic Tracing in a Case of Aneurysm opening into the Pulmonary Artery	413

NOTICE TO SUBSCRIBERS.

Terms of Subscription, including postage or delivery :

	s.	d.
In Great Britain, and to Countries within the Postal Union	6	0
To the Colonies, and to India	7	0
Terms to Non-subscribers	7	6

Subscriptions are due *immediately upon receipt of the volume*. Post-office orders should be drawn in favour of Dr. Frederick Taylor, made payable at the Borough High Street Post Office, and addressed to 11, St. Thomas's Street, Southwark, London, S.E. ; they may with advantage be crossed "and Co."

A printed and numbered receipt will in all cases (except for foreign Subscriptions) be sent to the Subscriber immediately on receipt of his remittance. If the Subscriber does not receive this within four days, he is requested to communicate at once with Mr. N. Davies-Colley, 36, Harley Street, W. In this way the Editors hope that all mistakes, of whatever kind, will be at once detected and investigated. The safe receipt of foreign remittances from countries within the Postal Union will be acknowledged by postal card. Changes of address, or any other corrections in the list of Subscribers, should be forwarded to the Editors.

It is not, however, necessary to notify to the Editors each year the Subscriber's wish to continue on the list, as no name will be erased so long as the volumes are duly paid for, unless at the express desire of the Subscriber.

In consequence of the regulations of the Postal Union, it is necessary to keep the weight of the volume below 2 lbs., so that it may readily be sent to countries within the Union.

NOTICE.

Somewhat imperfect sets of the First and Second Series of the Reports can be had at very reduced prices on application to the Editors.

LIST OF SUBSCRIBERS.

- Aberdeen, Medico-Chirurgical Society, The Library, Medical Hall, 29,
King Street, Aberdeen
Aberdeen University Library, Marischal College, Aberdeen
Abbott, George, 23, Finsbury Circus, E.C.
Adam, Mercer, M.D., The Churchyard, Boston, Lincolnshire
Adams, Matthew A., Ashford Road, Maidstone
Adeney, E. L., M.D., Bushey, Reigate
Aikin, Charles A., 7, Clifton Place, Sussex Square, W.
Aikins, M. H., M.D., Burnhamthorpe, Ontario, Canada
Alcock, Thomas, M.D., Oakfield, Ashton-upon-Mersey, Manchester
Anderson, G. E. C., Guy's Hospital
Andrews, Richard J., 19, Bedford Circus, Exeter
Ashby, Alfred, M.B., Grantham, Lincolnshire
Ashby, Henry, M.D., Guildhall Chambers, Lloyd Street, Albert Square,
Manchester
Atkins, F. D., High Street, Sutton, Surrey
Atkins, F. T., Wrottesley Lodge, Plumstead Common, Kent.
Atwell, Gregory H., Church Street, Altrincham, Cheshire
- Bader, C., 10, Finsbury Circus, E.C.
Bagnall, S. Freeman, 1, Clarence Terrace, Runcorn, Cheshire
Baker, A. de Winter, Dawlish, Devon
Baldwin, H. R., M.D., New Brunswick, New Jersey, United States of
America
Ball, J. A., M.B., Heaton-Norris, Stockport
Bankart, James, M.B., 19, Southernhay, Exeter
Banks, William A., M.D., Rockland, Maine, United States of America
Barrett, A. E., 12, Ladbroke Grove, W.
Barrett, J. W., Kings Lynn, Norfolk
Barrett, S. B. C., Kneller's Court, Fareham, Hampshire
Barrow, F. E., Surgeon with Royal Artillery, Tounghoo, Burmah,
Madras Presidency
Barra, A. G., M.D., 22, Park Place, Leeds
Barton, J. Kingston, 2, Courtfield Road, Gloucester Road, Queen's
Gate, S.W.
Batchelor, F. C., George Street, Dunedin, Otago, New Zealand
Beale, E. Clifford, M.B., B.A., 23, Upper Berkeley Street, Portman
Square, W.
Bealey, Adam, M.D., Oak Lee, Harrogate

- Beaney, The Hon. J. G., M.D., Collins Street, Melbourne, Australia
 Beardsley, Amos, Bay Villa, Grange-over-Sands, Lancashire
 Beddard, James, M.B., Park Row, Nottingham
 Beeby, Walter T., M.D., Bromley, Kent
 Berry, Henry Thomas, 29, Amwell Street, Claremont Square, E.C.
 Bettany, G. T., M.A., B.Sc., F.L.S., 2, Eckington Villas, Ashbourne
 Grove, East Dulwich
 Bevan, Richard, Cobb's Hall, Lydd, Kent
 Bird, Tom, M.A., 38, Brook Street, Grosvenor Square, W.
 Birdwood, R. A., B.A.
 Birkett, Edmund Lloyd, M.D., 48, Russell Square, W.C.
 Birkett, John, 59, Green Street, Grosvenor Square, W.
 Bisshopp, James, Bedford Place, Tunbridge Wells
 Blaker, Nathaniel P., 29, Old Steyne, Brighton
 Blaker, Thomas F. I., Avonhurst, Inholmes Park Road, Burgess Hill,
 Sussex
 Blason, Thomas, Billingborough, near Folkingham, Lincolnshire
 Booth, Lionel, M.D., Sherburn House, Durham
 Boswell, J. I., Faversham, Kent
 Bosworth, John Routledge, Sutton, Surrey
 Bovill, Edward, M.B., C.M., 32, James Street, Buckingham Gate,
 S.W.
 Bowen, A. L., 10, Lewisham Road, Blackheath, S.E.
 Bowen, Owen, 101, Heyworth Street, Everton, Liverpool
 Bowen-Jones, L. M., Whitland, R. S. O., Carmarthenshire
 Brailey, W. A., M.A., M.D., 16, Orchard Street, Portman Square, W.
 Branfoot, Henry S., M.B., 42, Norfolk Square, Brighton
 Bredin, R., M.B., 11, St. Edmond's Road, Bootle, Liverpool
 Brett, A. T., M.D., Watford, Hertfordshire
 Brett, J. T.
 Bright, John M., M.D., Alvaston, Park Hill, Forest Hill, S.E.
 Brogden, R. W., Guy's Hospital
 Brooks, Bransby, Sonning, near Reading, Berks
 Brown, Burton, M.D. (per F. G. Brown, Esq., 16, Finsbury Circus
 E.C.)
 Browne, George, 35, Montpelier Road, Brighton
 Bryant, Thomas, 53, Upper Brook Street, W.
 Buchanan, Walter, 2, Gibraltar Place, Chatham, Kent
 Budd, Herbert G., College Gates, Worcester
 Bunting, James, 1, York Villas, West Green Road, Tottenham
 Burdett, H. C., 39, Gloucester Road, Regent's Park, N.W.
 Burrell, Edwin, M.D., 20, Endsleigh Street, Tavistock Square, W.C.
 Burroughs, Thomas John, M.D., Crondall, Hampshire
 Burton, John M., Lee Park Lodge, Lee, S.E.
 Bushell, S. Wootton, M.D., Pekin, China (care of R. Mathews, Esq.)
 Butler, William Harris, 15, Thomas Street, Woolwich
 Button, Horace G., Southland Hospital, Invercargill, New Zealand
 (*via* San Francisco)
 Cann, Francis M., 6, Plantation Terrace, Dawlish, Devon

- Cardiff Medical Society (per T. G. Horder, Esq., 32, Crockherbtown, Cardiff)
- Carey, Francis, M.D., Villa Carey, Grange Road, Guernsey
- Carnelley, M., Guy's Hospital
- Carr, T., Richmond Terrace, Clayton, Manchester
- Carré, Louis C. A., 131, Camberwell Road, S.E.
- Carrington, R. E., M.D., 15, St. Thomas's Street, S.E.
- Carter, Thomas, Richmond, Yorkshire
- Cawley, Thomas, M.D., North Terrace, Adelaide, South Australia
- Champ, J. H., M.B., Guy's Hospital
- Cheese, James, 20, East Southernhay, Exeter
- Chicken, Rupert C., 54, Forest Road, Nottingham
- Clarke, Henry, H.M. Prison, Wakefield, Yorkshire
- Cleveland, W. F., M.D., Stuart Villa, 199, Maida Vale, W.
- Clifton Medical Reading Society (per Messrs. James Fawn & Son, Booksellers, 18, Queen's Road, Bristol)
- Clowes, Francis, Sutton Hall, Stalham, Norfolk
- Clowes, H. A.
- Clunn, T. R. H., County Asylum, Prestwich, Manchester
- Cock, Edward, Dean Street South, St. Thomas's Street, S.E.
- Cock, John, Exmouth, Devon
- Cock, Williams, 134, Queen's Road, Peckham, S.E.
- Cockell, Frederick Edgar, 268, Dalston Lane, Hackney, E.
- Cockell, Edgar, Holly Lodge, Forest Road, Dalston, E.
- Cogan, Lee F., 51, Sheep Street, Northampton
- Cole, R. M., Northgate House, Gloucester
- Collet, Aug. H., B.A., South Lodge, Grafton Road, Worthing, Sussex
- Collins, H. W., Wrington, near Bristol
- Collington, J. W., Kibworth, Leicestershire
- Colson, Edward, Port Surgeon, Steamer Point, Aden
- Cooke, James Wood, Barnstaple
- Cornwall, James, Fairford, Gloucestershire
- Couch, Thomas Quilter, Bodmin, Cornwall
- Couling, Henry, 11, Chesham Road, Brighton
- Court, Josiah, Staveley, Chesterfield
- Creed, C. P., Rathkeale House, Girdlers Road, West Kensington, W.
- Cregeen, J. Nelson, 93, Upper Parliament Street, Liverpool
- Crew, John, Higham-Ferrers, Northamptonshire
- Croft, John, 48, Brook Street, Grosvenor Square, W.
- Crompton, Dickinson W., 17, Temple Row, Birmingham
- Crooke, G. F., M.B., Fever Hospital, Leeds
- Crossley, C., 78, Granby Street, Leicester
- Cruise, F. R., M.D., 93, Merrion Square West, Dublin
- Cuff, R., M.B., 7, Huntriss Row, Scarborough
- Cunningham, John, M.B., Campbeltown, Argyleshire
- Currie, O. J., M.B., The Infirmary, Huddersfield
- Daglish, Richard Rothwell, Joy Cottage, New Romney, Kent
- Dakin, W. R., M.D., Guy's Hospital
- Daldy, Frederick Samuel, 2, Manor Place, Horsham, Sussex

- Dalton, B. N., M.D., Selhurst Road, South Norwood, S.E.
 Daniell, George Williamson, Blandford, Dorsetshire
 Darley-Hartley, W., East London, Cape of Good Hope
 Davies; Ebenezer, Brunswick House, Swansea
 Davies-Colley, J. N. C., M.A., M.C., 36, Harley Street, W.
 Davis, G., 11, The Avenue, Blackheath, S.E.
 Davy, Henry, M.D., 34, Southernhay, Exeter
 Debus, Henry, Ph.D., F.R.S., Athenæum Club, Pall Mall, S.W.
 Devon and Exeter Hospital Library (per James Bankart, Esq., M.B.,
 19, Southernhay, Exeter)
 Dix, John, 25, Albion Street, Hull
 Dodd, A. H., Guy's Hospital
 Dolman, A.H., 14, The Wardwick, Derby
 Douglas, W. T. P., B.A., M.B., Newbury, Berks
 Downes, G. L., Guy's Hospital
 Dryland, W., Dulwich Common
 Duke, Edgar, Freshwater, Isle of Wight
 Duke, Maurice S., 272, Kennington Park Road, S.E.
 Dunn, L. A., M.B., B.S., 26, Trinity Square, S.E.
 Duran, Carlos, and Nunez, Daniel, Costa Rica (care of Messrs. Le
 Lacheur and Son, 117 and 118, Leadenhall Street, E.C.)
 Durham, Arthur E., 82, Brook Street, Grosvenor Square, W.
 Durham, Frederic, M.B., 38, Brook Street, Grosvenor Square, W.
 Dutton, Edward G.
- Eager, Reginald, M.D., Northwoods Asylum, Frampton Cotterell, near
 Bristol
 Eastes, George, M.B., 69, Connaught Street, Hyde Park Square, W.
 Eastes, Thomas, M.D., 3, Shakespeare Terrace, Folkestone
 Edwards, O., South Street, Leominster, Herefordshire
 Elder, George, M.B., C.M., 17, Regent Street, Nottingham
 Elphick, Edward, Tea Tree Gully, near Adelaide, South Australia
 Elphinstone, Robert, 46, St. John's Wood Park, N.W.
 English, D.C., M.D., New Brunswick, New Jersey, United States of
 America
 Evans, Alfred H., Sutton Coldfield, Warwickshire
 Evans, John Henry, 33, Sandy Road, Seaforth, Liverpool
 Evershed, Arthur, M.R.C.P. Lond., 10, Mansfield Villas, Hampstead,
 N.W.
 Evershed, Charles L., Arundel, Sussex
 Ewart, J. H., Eastney, Devonshire Place, Eastbourne
 Ewen, H. W., care of A. Kelsey, Esq., Station Road, Redhill, Surrey
- Faircloth, Richard, 3, Inverness Gardens, Campden Hill, Kensing-
 ton, W.
 Farr, George F., Slade House, Kennington Road, S.E.
 Fawsitt, Thomas, 46, Union Street West, Oldham
 Fergusson, James, M.D., Strathalbyn, South Australia
 Few, William, Ramsey, Huntingdon
 Field, Ernest, M.D., C.M., 12, Queen Square, Bath

- Forster, J. Cooper, 29, Upper Grosvenor Street, W.
Forty, D. H., The Rectory, Wootton-under-Edge, Gloucestershire
Foster, F. W., Guy's Hospital
Foster, O. H., M.A., M.B., Hitchin, Hertfordshire
Fotherby, Henry I., M.D., 3, Finsbury Square, E.C.
Fowler, George, 170, Kennington Park Road, S.E.
Fowler, W., M.B., Guy's Hospital
Fry, J. F., Belvoir, St. Helen's Road, Swansea, South Wales
Fuller, Thomas, M.D., Longcrofts, New Shoreham, Sussex
- Galabin, A. L., M.A., M.D., 49, Wimpole Street, W.
Galton, John H., M.D., 39, Anerley Road, Upper Norwood, S.E.
Galton, E. H., Brixton Rise, Surrey, S.W.
Gardner, J. T., 6, Hillsboro' Terrace, Ilfracombe, Devon
Garrard, C. R. O., Rotherham Hospital, Rotherham, Yorkshire
Garrard, W. A., Wellgate, Rotherham, Yorkshire
Gathergood, B. W., Terrington St. John, Lynn, Norfolk
Gay, W., St. James's Lodge, Doncaster
Golding-Bird, C. H., B.A., M.B., 13, St. Thomas's Street, S.E.
Goodhart, J. F., M.D., 25, Weymouth Street, Portland Place, W.
Gorham, John, Tunbridge, Kent (through Bookseller)
Gosse, H. W., Midsomer-Norton, Bath, Somerset
Gowing, Benjamin C., Avon House, Salisbury
Goyder, David, M.D., 88, Great Horton Road, Bradford, Yorkshire
Graham, John, M.D., Bryckden, Waldron, Sussex
Greenwood, E. C., Guy's Hospital
Griffiths, Owen
Gross, Charles, Newington Infirmary, Westmoreland Road, Walworth, S.E.
Grove, W. R., M.D., St. Ives, Huntingdonshire
Groves, Charles E., F.R.S., Kennington Green, S.E.
Growse, J. L., Bildeston, Suffolk
Growse, W., Guy's Hospital
Gull, Sir W. W., Bart., M.D., D.C.L., F.R.S., 74, Brook Street, Grosvenor Square, W.
Guy, Thomas, M.D.
Guy's Hospital Library (Two Copies)
- Habershon, S. O., M.D., 70, Brook Street, Grosvenor Square, W.
Haden, W. H., M.D., 66, Harley Street, W.
Hall, James Griffith, J.P., Swansea
Halls, Thomas Edward, 64, Tooley Street, S.E.
Hants Royal County Hospital, Winchester
Harding, C. F., M.D., Whittlesea, Cambridge
Harrinson, Isaac, Castle Street, Reading
Harris, Robert, M.B., 10, Walmer Road, Birkdale, Southport
Harris, Vincent D., M.D., 39, Wimpole Street, Cavendish Square, W.
Harrison, A. J., M.B., Failand Lodge, Guthrie Road, Clifton
Hartree, J. P., M.A., M.B., Leigh, Tunbridge
Harvey, C. T., 42, Hoghton Street, Southport

- Hayward, John W., Whitstable, Kent
 Heddy, William Jackson, 25, Hollywood Road, South Kensington, S.W.
 Hedley, Charles, Welford, Rugby
 Hibberd, E., M.D., Campfield Lodge, Walterton Road, St. Peter's Park, W.
 Hicks, John Braxton, M.D., F.R.S., 24, George Street, Hanover Square, W.
 Higgins, C., 38, Brook Street, Grosvenor Square, W.
 Higgins, Charles Hayes, M.D., Alfred House, Birkenhead, Cheshire
 Hills, A. Phillips, Carlton House, Bridge Road, Battersea Park, S.W.
 Hills, William Charles, M.D., County Lunatic Asylum, Thorpe, Norwich
 Hindle, F. T., Askern Hill, near Doncaster
 Hobson, J. M., M.D., 3, Addiscombe Villas, Croydon
 Hodson, Frederic, Hornsea, Hull
 Holman, Constantine, M.D., J.P., Reigate, Surrey
 Hood, Donald W. Charles, M.D., 43, Green Street, Park Lane, W.
 Horrocks, Peter, M.D., 9, St. Thomas's Street, S.E.
 Houseman, John, M.D., 68, Jesmond Road, Newcastle-upon-Tyne
 Howard, Dr., 47, Union Avenue, Montreal, Canada (per Mr. H. Kimpton, 182, High Holborn)
 Howell, J. B., The Old Vicarage, Wandsworth, S.W.
 Howell, T. A. I., The Old Vicarage, Wandsworth, S.W.
 Howse, H. Greenway, M.S., 10, St. Thomas's Street, Southwark
 Hubbard, Frank E., Kent and Canterbury Hospital, Canterbury
 Hudson, R. S., M.D., Redruth, Cornwall
 Hughes, Robert Harry, M.A., M.B., 12, Lockyer Street, Plymouth
 Hutchinson, V., M.D., The Elms, Bishop Auckland, Durham

 Ince, John, M.D. (per Messrs. Grindlay and Co.)

 Jackson, A. C., Cape Town, South Africa
 Jackson, Arthur, Wilkinson Street, Sheffield
 Jackson, P. J., Surrey Dispensary, 6, Great Dover Street, S.E.
 Jacobson, W. H. A., B.A., M.B., 41, Finsbury Square, E.C.
 Jalland, W. H., St. Leonard's House Museum Street, York
 James, Philip, Pandy House, Llwyn-y-pia, Pont-y-Pridd, Glamorgan-shire
 James, W. C., M.D., 11, Marloes Road, Cromwell Road, Kensington, W.
 Jennings, Oscar, M.D., 43, Boulevard Malesherbes, Paris
 John, Wm., Court House, Haverfordwest, Pembrokeshire
 Johnson, David, M.D., Manor House, 164, Earl's Court, South Kensington, S.W.
 Johnson, W. F., Charing, near Ashford, Kent
 Jones, A. H., M.D., 32, Sheep Street, Northampton
 Jones, George, B.A., Framlingham, Suffolk
 Jones, J. Edwards, M.D., Brynffynon, Dolgelly, North Wales
 Jones, John Thomas, 179, Brixton Road, S.W., and Llanfyllin, Montgomeryshire

Jones, Morris, Aberystwith, Cardiganshire
Jones, Thomas, M.B., 96, Mosley Street, Manchester
Jones, W. Makeig, Wath-upon-Deane, Rotherham
Joyce, Thomas, 2, Pembridge Gardens, Bayswater, W.
Judson, T. R., West Derby, Liverpool

Keep, Charles H. (care of Dr. Willett, Wyke House, Syon Hill, Isleworth)

Kellock, W. B., Stamford Hill, Stoke Newington, N.
Kelsey, A., Station Road, Redhill, Surrey
Kelso Dispensary, Roxburghshire (per Dr. Thomas Hamilton)
Kendall, Walter B., 5, South Terrace, Dorchester
Kent, Thomas J., 89, Piccadilly, W.
Ker, Hugh Richard, Townsend House, Hales-Owen, Worcestershire
Kidd, W. A., M.D., B.S., 12, Montpelier Row, Blackheath, S.E.
Kingsford, Edward, Sunbury, Middlesex

Lacey, John, 23, Trinity Street, Southwark, S.E.
Lacey, T. W., 196, Burrage Road, Plumstead
Lacy, A. G., The Cottage, Sunninghill, Ascot, Middlesex
Lamb, Joseph, 18, Price Street, Birkenhead
Lamb, William Henry, M.B., 46, Kensington Park Gardens, W.
Lancereaux, E., M.D., 3, Rue St. Arnaud, Paris
Lane, W. A., M.S., 14, St. Thomas's Street, S.E.
Lansdown, F. P., 19, White Ladies' Road, Clifton, Bristol
Larkin, F. G., Grove Park, Lee, Kent
Lee, C. G., 84, Bedford Street South, Liverpool
Leeds School of Medicine Library (per Dr. Allbutt, School of Medicine, Leeds)
Lewis, Charles, 67, Sandgate Road, Folkestone
Lipscomb, John Thomas N., M.D., St. Albans, Hertfordshire
Lister, J. H., 23, Hunter Street, Bedford Square, W.C.
Littlewood, J. O.
Love, Augustus E. B., Richmond Villa, Bournemouth
Lucas, R. Clement, B.S., 18, Finsbury Square, E.C.
Lucas, Herbert, Huntingdon
Lund, Edward, 22, St. John Street, Manchester
Lund, H., Guy's Hospital
Lush, Wm. George Vawdrey, M.D., 12, Frederick Place, Weymouth

McKay, W. W., M.D., Main Street, Boise City, Idaho Territory, United States of America

Mackenzie, Morell, M.D., 19, Harley Street, W.
Mackern, George, M.D., 208, Calle Generale Lanalle, Buenos Ayres
Mackie, J., M.D.
Maconchy, John K., M.B., Infirmary House, Downpatrick
Madge, Henry, M.D., 4, Upper Wimpole Street, W.
Mahomed, F. A., M.D., 24, Manchester Square, W.
Makens, J., M.B.
Mallam, G. B.

- Mallam, W. P., 169, Uxbridge Road, Shepherd's Bush, W.
 Manby, Frederic, East Rudham, Swaffham, Norfolk
 Manby, Frederic E., 10, King Street, Wolverhampton
 Manchester Royal Infirmary (per The Secretary)
 Mansell, E. R., 6, Belle Vue, West Hill, Hastings
 Marshall, Edward, Mitcham, Surrey
 Martin, A. Rae, The Precincts, Rochester, Kent
 Martin, F., M.D., Holmhurst, Southend Road, Beckenham, Kent
 Martin, Joseph Cooper, 2, West Hill, Dartford, Kent
 Masters, J. A., Westall House, Brook Green, West Kensington, W.
 Mathews, Robert, Bickley, Kent
 Mead, H. R., Fishponds, near Bristol
 Mickley, Arthur G., M.B., Buntingford, Herts
 Mickley, George, M.B., M.C., St. Luke's Hospital, Old Street, E.C.
 Milligan, R. A., 39, Sheep Street, Northampton
 Milward, James, 54, Charles Street, Cardiff
 Moon, Henry, 26, Finsbury Square, E.C.
 Moon, R. H., Fern Lodge, Lower Norwood, Surrey
 Moore, J., M.D.
 Morgan, David C.
 Morgan, John, Pontrhyd-y-groes, near Aberystwith
 Morison, Joshua W., Hamilton Terrace, Pembroke, South Wales
 Morley, Edward S., M.D., 16, Richmond Terrace, Blackburn
 Morley, J. L. Collison, M.D., 124, Edith Road, West Kensington, W.
 Morris, Henry, M.A., M.B., 2, Mansfield Street, Portland Place, W.
 Morris, John, 30, Dorset Gardens, Brighton
 Morse, R. E. R., Eton House, Oriel Terrace, Cheltenham
 Moxon, H. J.
 Moxon, Walter, M.D., 6, Finsbury Circus, E.C.
 Munden, Charles, Ilminster, Somerset
 Muriel, Charles Evans, 71, St. Giles's Street, Norwich
 Muriel, George John, 14, Scotch Street, Whitehaven
 Murphy, S. F., 158, Camden Road, N.W.
- Nason, John James, M.B., Stratford-on-Avon
 Newman, Alfred K., M.B., The Club, Wellington, New Zealand
 Newman, J. J., 6, Matlock Terrace, Torquay
 Nisbett, Robert Innes, The Eagles, Overcliff, Gravesend
 Northampton General Infirmary Library (per the House Surgeon)
 Nowell, R. B., Henry Square, Ashton-under-Lyne
 Nunn, George Richard, Lyndhurst, Hampshire
 Nunneley, John, M.B., 22, Park Place, Leeds
- O'Grady, E.S., M.B., C.M., 105, Stephen's Green South, Dublin
 Oldham, Henry, M.D., 4, Cavendish Place, W.
 Oxley, W., 1, South Terrace, Rotherham, Yorkshire
- Padley, George, Northampton Lodge, Swansea
 Palmer, W. G., Loughborough, Leicestershire

- Paramore, Richard, 18, Hunter Street, Brunswick Square, W.C.
Parker, Robert W., 8, Old Cavendish Street, Cavendish Square, W.
Parkinson, C. H. W., Wimborne-Minster, Dorset
Parry, Robert, M.B., Carnarvon
Paul, Frank T., 44, Rodney Street, Liverpool
Pavy, F. W., M.D., F.R.S., 35, Grosvenor Street, W.
Payne, Arthur J., M.D., Surgeon-Major, Bengal Medical Service (per Messrs. Lewis & Co., Gower Street)
Pearse, E. Sainthill, 3, Albion Street, Brierley Hill, Staffordshire
Peat, Thomas, Manningtree, Essex
Pegge, Charles, Vernon House Asylum, Briton Ferry, Glamorganshire
Perkins, Charles E. S., M.B., 2, Kirkdale, Sydenham, S.E.
Perks, R. H., Guy's Hospital
Phillipps, W. A., 4, Court Street, Faversham, Kent
Phillips, H. Astley, M.B., C.M., Denny House, Waterbeach, Cambridgeshire
Pilcher, W. J., High Street, Boston, Lincolnshire
Pilkington, F. S., Guy's Hospital
Pilkington, F. W., Chorley, Lancashire
Pilkington, George, Yarm-on-Tees, Yorkshire
Pinching, Charles J. W., 76, New Road, Gravesend
Pitt, G. N., M.A., M.D., 34, Ashburn Place, South Kensington
Plimmer, H. G., Brantwood, Waldegrave Road, Upper Norwood, S.E.
Plomley, John Fred., M.D., 9, West Boro,' Maidstone
Poland, John, 27A, Finsbury Square, E.C.
Portman Medical Book Club (per E. Owen, Esq., 49, Seymour Street, Portman Square, W.)
Prall, S. E., Guy's Hospital
Prance, R. R., M.D., Rookeslea, Greenhill Road, Hampstead, N.W.
Prendergast, J. J., Guy's Hospital
Price, J. A. P., M.B., Reading
Purdon, T. H., M.B., 5, Wellington Place, Belfast
Purves, Laidlaw, 6, Stratford Place, Oxford Street, W.
Puzey, Chauncy, 71, Rodney Street, Liverpool
Pye-Smith, E., St. Catharine's Lodge, Sevenoaks, Kent
Pye-Smith, Philip H., M.D., 54, Harley Street, W.
Pye-Smith, R. J., 6, Surrey Street, Sheffield
- Rake, B. N., M.D., Government Medical Officer, Peru House
Mucurapo, Port of Spain, Trinidad
Ramskill, Josiah, 29, Meadow Lane, Leeds
Ramskill, J. S., M.D., 5, St. Helen's Place, Bishopsgate, E.C.
Rand, John, Walton House, Grove Hill, Dulwich, S.E.
Ray, Edward Reynolds, North Dulwich, S.E.
Rees, G. Owen, M.D., F.R.S., 26, Albemarle Street, W.
Reinold, A. W., M.A., F.R.S., Royal Naval College, Greenwich, S.E.
Rendle, Richard, Treverbyn, Dartmouth Park, Forest Hill, S.E.
Reynolds, L. W., High Wycombe, Bucks
Reynolds, W. P., Stamford Hill, N.
Richardson, H. E., Borough Hospital, Birkenhead

- Richardson, T. A., 24, London Road, West Croydon
 Richmond Hospital Library, Dublin (per Dr. Gordon)
 Roberts, Alfred, 45, Philip Street, Sydney, New South Wales
 Roberts, Bransby, M.D., Badlesmere House, Eastbourne, Sussex
 Roberts, H. J., Guy's Hospital
 Roberts, J. H., Hill Crest, Greenhill Road, Hampstead, N.W.
 Rogers, Robert J., 40, Cannon Place, Brighton
 Romano, F. W. R., Pelotas, Rio Grand do Sul, Brazil
 Rooke, Thomas Morley, M.D., 7, Bays Hill Villas, Cheltenham
 Roots, W. Henry, Kingston-on-Thames, Surrey
 Roper, A. G., 57, North End, Croydon
 Roper, Arthur, 17, Granville Park, Blackheath
 Roper, E., Highfield House, Shepperton, Middlesex
 Ross, John Harris, M.D., C.M., 8, St. George's Place, Brighton
 Ross, Richard, M.D., 7, Wellington Place, Belfast
 Ryle, R. J., Guy's Hospital

 Salter, S. J. A., M.B., F.R.S., 49, Devonshire Street, Portland
 Place, W.
 Salzmann, Frederick William, 18, Montpellier Road, Brighton
 St. Mary's Hospital, Manchester (care of the Librarian)
 Sanders, J. W., Guy's Hospital
 Sangster, Charles, 148, Lambeth Road, S.E.
 Savage, G. H., M.D., Bethlem Royal Hospital, S.E.
 Scott, Francis, Addison House, Edmonton, Middlesex
 Scott, R. J. H., 13, Bladud Buildings, Bath
 Sells, C. J., White Hall, Guildford
 Sharp, John Adolphus, 61, Osmaston Street, Derby
 Shaw, C. T. Knox, 33, Warrior Square, St. Leonard's-on-Sea
 Sheldon, T. Steele, M.B., Somerset and Bath Lunatic Asylum, Wells
 Shelswell, O. B., Holborn Union Infirmary, Archway Road, Upper
 Holloway
 Shepherd, A. B., M.A., M.D., 17, Great Cumberland Place, Hyde
 Park, W. (for the Library of the University of Freiburg, in Baden)
 Shipman, George Wm., Grantham, Lincolnshire
 Sigler, Geo. A., M.D., Liberty, Union County, Indiana, United States
 of America
 Simon, R. M., B.A., M.B., 27, Newhall Street, Birmingham
 Skinner, David S., M.D., 1, Bedford Gardens, Campden Hill, W.
 Skinner, William A., 45, Lower Belgrave Street, S.W.
 Smallpeice, W. D., 42, Queen Anne's Gate, St. James's Park, S.W.
 Smith, J. S., Truro Road, St. Austell, Cornwall
 Smith, James William, 13, Hall Gate, Doncaster
 Spender, J. K., M.D., 17, Circus, Bath
 Spry, G. Frederick Hume, M.D., 2nd Life Guards, Army and Navy
 Club, Pall Mall, S.W.
 Spurgin, Herbert B., 45, Abington Street, Northampton
 Spurgin, Thomas, Manor House, Ongar, Essex
 Stamper, James F., M.D., Pembroke Dock, South Wales
 Starling, E. A., Coventry and Warwickshire Hospital, Coventry

- Stedman, John B., Godalming, Surrey
Steele, J. C., M.D., Guy's Hospital
Steele, Richard, Great Peatling, Lutterworth, Leicestershire
Stephens, Thomas Palmer, Westbourne, Sussex
Stevenson, Thomas, M.D., Sandhurst Lodge, Gresham Road, Brixton
S.W.
Stilwell, Robert R., M.D., Beckenham, Kent
Stocker, John Sherwood, M.D., 2, Montagu Square, W.
Stoke Newington, Clapton and Hackney Medical Book Society (per
F. Wallace, Esq., 96, Cazenove Road, Upper Clapton)
Stothard, W. J., Sandburne, Palatine Road, Didsbury, Manchester
Stuart, E. O., Hope Hospital, Eccles, Manchester
Sturge, H. H.
Sutton, Frederick, Willingham-by-Stow, Gainsboro', Lincolnshire
Sutton, Henry G., M.B., 9, Finsbury Square, E.C.
Sydney-Turner, A. M., College Green, Gloucester
Symonds, Charters James, M.S., 16, St. Thomas's Street, S.E.
- Taylor, Frederick, M.D., 11, St. Thomas's Street, S.E.
Taylor, R. Stanley, M.B., 7, Friar Gate, Derby
Taylor, Thomas, Sutton Coldfield, Warwickshire
Ticehurst, C. S., Petersfield, Hants
Ticehurst, Augustus R., Silchester House, Pevensey Road, St.
Leonards-on-Sea
Todd, J., 20, King Street, Lancaster
Townsend, T. S., 68, Queen's Gate, South Kensington, S.W.
Trevor, E. T., Guy's Hospital
Tubby, A. H., Guy's Hospital
Tuchmann, M., M.D., 148, Adelaide Road, Haverstock Hill, N.W.
Turner, H. G., Holmwood, Bournemouth
Tyson, W. J., M.D., 10, Langhorne Gardens, Folkestone
- Uhthoff, J. C., M.D., 46, Western Road, Hove, Brighton
Underhill, W. Lees, Tipton Green, Staffordshire
- Valentine, Edmund William, Somerton, Somersetshire
Vawdrey, George, Clifford House, Shaldon, Teignmouth, Devon
Veasey, Henry, Woburn, Bedfordshire
Vesey, T. A., M.B., Knapton, Rosstrevor, Ireland
- Wacher, Frank, Kingsbridge, Canterbury
Waddy, H. E., 30, Clarence Street, Gloucester
Wainwright, Robert S., M.D., Belmont, Lee, Kent, S.E.
Wales, T. Garneys, Downham Market, Norfolk
Walker, C. P., Guy's Hospital
Wallace, Frederick, 96, Cazenove Road, Upper Clapton, E.
Wallace, Richard U., M.B., 186, Amhurst Road, Hackney Downs, E.
Waller, W. A. E., 46, Albert Street, Rugby
Wallis, W., jun., Groombridge, Tunbridge Wells
Warner, P., Oaklands, Woodford, Essex

- Washbourn, Buchanan, M.D., Gloucester
 Watson, W., Theobald House, Rochester, Kent
 Weaver, F. P., M.D., Frodsham, Cheshire
 Weber, Hermann, M.D., 10, Grosvenor Street, W.
 Weir, Patrick A., M.A., M.B., C.M., Surgeon Bengal Army (per
 Messrs. King, Hamilton, and Co., 7, Hare Street, Calcutta)
 West-Jones, G. H., Eckington, Chesterfield, Derbyshire
 Weston, E. F., The Green Hall, Stafford
 Wheeler, D. M. B., Chelmsford
 White, B. P., M.D., Pump Street, Londonderry
 White, Charles, Warrington
 White, R. W., 25, Broad Street, Leominster
 White, W. Hale, M.D., 4, St. Thomas's Street, S.E.
 Wilkin, J. F., M.D., M.C., The Warren, Beckenham Park, Kent
 Wilks, Samuel, M.D., F.R.S., 72, Grosvenor Street, Grosvenor
 Square, W.
 Willan, G. T., Melton Mowbray, Leicestershire
 Willett, Edmund S., M.D., Wyke House, Syon Hill, Isleworth
 Williams, W. R., M.D., Commission on Lunacy Office, 19, Whitehall
 Place, S.W.
 Williamson, N., M.D., New Brunswick, New Jersey, United States of
 America
 Wilton, J., Chalk-pit House, Sutton, Surrey
 Wise, William C., M.D., Gothic Villa, Burrage Road, Plumstead, Kent
 Wiseman, John Greaves, Dearden Street, Ossett, Wakefield
 Wolstenholme, H. J., High Cross, Tottenham
 Wood, P. M., Yam Creek, Port Darwin, Australia (viâ Hongkong)
 Wordley, A. W., 8, Great Suffolk Street, S.E.
 Wright, Charles J., Lynton Villa, Virginia Road, Leeds
 Wright, G. A., B.A., M.B., 8A, St. John Street, Manchester

 York Medical Society (care of Fred. Shann, Esq., 69, Petergate,
 York)

IN EXCHANGE.

The St. Bartholomew's Hospital Reports.

The St. George's Hospital Reports.

The Liverpool and Manchester Medical and Surgical Reports.

The St. Thomas's Hospital Reports.

The Pharmaceutical Journal.

The Transactions of the Obstetrical Society of London.

American Journal of the Medical Sciences (care of Messrs. Trübner and Co., Ludgate Hill, E.C.).

Annales de Dermatologie et de Syphiligraphie (care of M. le Dr. Ernest Besnier, 87, Rue Neuve des Mathurins, Paris).

Journal de Théraputique (care of M. le Dr. Labbée, 10, Rue de Turbigo, Paris).

Revue des Sciences Médicales en France, et à l'Étranger (care of M. le Dr. Hayem, 17, Rue du Sommerard, Paris).

Verhandlungen der Berliner medicinischen Gesellschaft (care of Herr A. Falk, Library Gutmann, Friedrichs Strasse 97, Berlin).

Centralblatt für Chirurgie (care of Messrs. Breitkopf und Härtel, Leipzig).

Beiträge zur Medizinal-Statistik (care of Dr. W. Zuelzer, Wilhelm Strasse 68, Berlin, W.).

Upsala Läkareförenings Förhandlingar (per Prof. Hedenius, Bibliothèque de la Société des Médecins, Upsal, Suède).

Le Progrès Médical (per Dr. Bourneville, Rue des Écoles 6, Paris).

Chicago Medical Journal and Examiner (per Dr. W. H. Byford, care of Messrs. W. B. Keen, Cooke, & Co., 113 and 115, State Street, Chicago, United States of America).

Transactions of the American Medical Association (per Messrs. Churchill).

Transactions of the American Gynæcological Society, Clarendon Street, Boston, Mass. (per Messrs. Churchill).

Library of Surgeon-General's Office, U.S. Army, Washington, D.C. (per Mr. B. F. Stevens, U.S. Government Despatch Agency, 4, Trafalgar Square, London, W.C.)

Transactions of the Brooklyn Anatomical and Surgical Society, 28, Madison Street, Brooklyn, New York, U.S.A.

The Sanitary Record, 15, Waterloo Place, S.W.

Mémoires de la Société de Médecine et de Chirurgie de Bordeaux (per Dr. Demons, Hôpital St. André, Bordeaux).

In Memoriam.

CHARLES HILTON FAGGE.

CHARLES HILTON FAGGE was born at Hythe on June 30th, 1838, and died at Grosvenor Street, November 18th, 1883. He was the son of Mr. Charles Fagge, who is still living at Hythe, where he has been engaged many years in general practice. His mother was the sister of the late distinguished surgeon of Guy's Hospital, John Hilton, after whom the subject of this memoir was named. Hilton Fagge was educated first at a private school near Brentwood, subsequently at Blackheath. He was so successful in his classical and mathematical studies that the masters advised his father to send him to one of the older Universities. Circumstances, however, prevented this course, and in October, 1856, when he was eighteen years of age, he entered as a student at Guy's Hospital. Being of a retiring and a modest temper he did not make his presence at once known, more especially as at that time the prizes for merit on special subjects of medical study had been abolished and the present system of awarding scholarships to the best men of the year had not come into vogue. He therefore had no opportunity of openly distinguishing himself amongst his fellows, but fortunately he found a larger competitive field at the London University, where candidates congregate from all the schools in the United Kingdom. Here his superiority was at once displayed, leading to a success so remarkable as to constitute a unique example in the annals

of the University. It became then apparent that Fagge had remarkable talents, marked more especially by the possession of a good memory, indefatigable industry, and by that which was his most characteristic feature, a faculty of acute perception whereby he could grasp all the salient points of a subject, enabling him to hold firmly to all that was true and important whilst he could dismiss the useless and false.

At the Matriculation Examination of the London University in 1857 he gained the first prize in Botany and the second in Chemistry, and in the following year he obtained the medal in Botany given by the Apothecaries' Company.

At the first M.B. examination in 1858 he gained the scholarship and gold medal in Anatomy and Physiology; he obtained the same in Materia Medica and the same in Chemistry, as well as a gold medal in Botany.

At the second M.B. examination, in 1861, he obtained the scholarship and gold medal in Medicine, the same in Physiology and a gold medal in Surgery. He stood also second in Midwifery.

In 1853 he took his M.D. degree, in 1864 became a Member of the Royal College of Physicians, and in 1870 a Fellow of the same learned body.

On the first vacancy occurring at Guy's Hospital, Fagge was appointed Demonstrator of Anatomy; in 1866 he became Medical Registrar, and on the death of Dr. Barlow, in 1867, was elected Assistant Physician, and took charge of the out-patient Skin Department. He subsequently joined Dr. Moxon as Demonstrator of Morbid Anatomy and two years later succeeded him as Curator of the Museum. In 1880 he was elected full Physician to the hospital. Amongst other appointments he held that of Physician to the Infirmary for Children and Women, and was also Physician to the Evelina Hospital. He was for some years Editor of the 'Guy's Hospital Reports,' and at the time of his death was Examiner in Medicine at the University of London.

During the time that Dr. Fagge was holding these various appointments he was assiduously working towards the advancement of medicine. He was essentially a clinical physician or pathologist in its widest and largest sense. He showed no predilection for any one class of disease, which indicates often some mental peculiarity on the part of its possessor, urging

him towards speculative inquiry, on the one hand, or leading him, on the other, to some inferior work requiring little more than mechanical nicety for its aim. Fagge could not thus narrow himself to a special inquiry, possessing as he did the faculty of grasping large and important facts of all kinds, analysing them and grouping them in such a manner as to make many subjects which were before obscure stand out in a newer and clearer light. His true place was in the wards searching for facts, making his morbid anatomy and chemistry subservient to the solution of larger questions of clinical medicine or pathology. In his remarkable power of insight into obscure forms of disease, and of discovering an explanation of symptoms, he possessed the talents of his uncle. Whatever Mr. Hilton touched he adorned, and however commonplace the subject for consultation, were it only a fractured leg, an ulcer, or hæmorrhoids, he would discover some new point in its character or discuss its scientific treatment in so lucid a manner that he would leave it surrounded by a halo of interest. Not long before Mr. Hilton's death the writer met him at the bedside of a friend who was suffering from a diffuse cellular inflammation of the neck and throat, a well-known and long recognised malady but lately honoured with the grandiose name of "angina Ludovici." Mr. Hilton, finding no mechanical impediment in the larynx to justify an operation, turned to the medical man present to ask what then was the cause of the great and peculiar difficulty of breathing; on having no response he answered himself by saying, "the implication of the pneumogastric nerves in the inflammatory process." Thus he was ever ready to throw light by his sagacity and knowledge on what was before obscure. His nephew possessed much of the same faculty of discernment.

At the very onset of Hilton Fagge's career he studied under Hebra, and interested himself in cutaneous diseases. He accordingly undertook the translation of some of the volumes of the Vienna professor for the New Sydenham Society. The work was done in the best and most conscientious manner, as the correspondence between author and translator proves. All the doubtful passages were discussed between them, and Hebra finally offered a just tribute of praise to his coadjutor. Dr. Fagge afterwards rearranged and classified the wax models of

skin disease in the museum according to the more advanced nomenclature,¹ and added to their value by the publication of a well written catalogue. One of Fagge's most interesting and original papers had reference to skin diseases. In his essay on "Scleriosis and Allied Affections" in the 'Guy's Hospital Reports' he brought together a number of cutaneous affections which had before been isolated or confounded, such as keloid, morphea, and various supposed forms of lepra. These he analysed and grouped, producing a rational, scientific, and what is now a classical paper. He also investigated afresh the subject of vitiligoidea, already treated of in a former volume of these Reports by Addison and Gull; and by a much more extended inquiry he showed, what has subsequently been confirmed, that the subjects of this disease are mostly affected by hepatic disorders. Before this, however, he had met with a good case of molluscum fibrosum, and he did not lose the opportunity of searching out its nature. The result of his work he took to the Royal Medical and Chirurgical Society, and showed drawings, which were then novel, of the development of the tumours in the hair follicles and sebaceous glands. Other papers on various kinds of diseases of the skin and nails are to be found in the 'Guy's Hospital Reports.'

Dr. Fagge's characteristic abilities have already been alluded to, his remarkable perspicuity, and his clear mental vision, which enabled him to separate the true from the false, the wheat from the chaff, and to perceive the relation which his facts had to one another, so as to dissect or unite them, as the case might be, in order to build up a new piece of pathology. He could generalise or even frame a theory, but he would never theorise without some good basis, or speculate without some show of reason. His turn of mind was a complete antidote to all that fanciful and flighty writing which emanates from the author's

¹ The excellent collection of wax models of skin disease made by the late Mr. Towne was commenced at the instigation of Addison. Addison had been a pupil of Bateman, and probably knew more of cutaneous affections than any other physician of his time. He continued, however, to use the terms of his master, and the wax models were labelled according to Willan and Bateman's nomenclature. Not long before Addison's death Hebra paid a visit to the Museum, and was much struck by the beauty and accuracy of the models, but on leaving the place he exclaimed to his attendant, "The artist seems able to recognise the diseases and to copy them, but the physician does not know what to call the models when they are made."

brain when surrounded by books in his study. Fagge's place was in the wards, and his mind was practical; and, although he probably possessed as much literary knowledge of medicine as any man of his day, being a good French and German scholar, he was able to temper and correct all he read by his own experience, allowing nothing to emanate from him that he had not well assimilated. Probably nothing that he has written will ever be gainsaid or found erroneous. His labour was that of assisting in building up the edifice of medicine, and to this he has added stones which will for ever endure.

No advantage will be gained by dwelling further on Fagge's peculiar abilities; it were better to offer as illustrations of his power a few more of his contributions to medicine, and record them here in our hospital archives.

Dr. Fagge mentions in the MS. of the work which is now in course of publication that on his first introduction to Guy's Hospital by his father, he went into the post-mortem room and there saw a case of aneurysm of the heart. The writer of this notice well remembers the circumstance, and he has thought it swayed Fagge's mind towards an interest in heart disease. Certainly some of his most elaborate contributions to medical literature were on this subject. His article on "Valvular Disease of the Heart" in 'Reynolds' System of Medicine' is a masterly one; in this article, and in a previous unsigned communication to the 'Brit. and For. Med.-Chirurgical Review,' Fagge was the first in this country to give the new views of Chauveau as to the causation of bruits, and he handled the subject in a most logical and scientific spirit. His paper also on the "Heart" in connection with presystolic bruit and contracted mitral in the 'Guy's Hospital Reports,' was one of special and novel interest at the time it was written. Laennec had been forgotten and Gairdner had revived or rediscovered the facts which Fagge helped to elucidate by at once taking up what was evidently to him a most important addition to the clinical knowledge of cardiac disease. Another paper, which may fairly be called classical, is that on "Fibroid Disease of the Heart" in the 'Transactions of the Pathological Society.' He collected a large number of cases exhibiting this condition, pointed out its connection with pericarditis and syphilis, and showed how often it was one associated with sudden death.

Dr. Fagge had previously contributed to the Society a paper on "Endocarditis in Pyæmia," the object being to show how, pathologically as well as clinically, these two affections had much in common. He had also reported a case of dissecting aneurysm in the 'Transactions' of the Royal Medical and Chirurgical Society, in order to show how in the more chronic forms, where pouches sometimes occur in the external coat, they become lined by a smooth membrane. The first paper which Fagge wrote when a pupil proved that he did not intend any subject of interest to pass his notice. He observed a case on the post-mortem table where there was an abnormal arrangement of the femoral artery; he dissected the limbs and discussed the irregularity in connection with the development of the circulatory system. On an allied subject, the action of digitalis on the heart, he made elaborate experiments in conjunction with Dr. Stevenson, the result of which was in part sent to the Royal Society, and more fully described in the 'Guy's Hospital Reports' of 1866. Besides the full account of the effects of the drug, the authors insisted strongly upon the value of physiological experiments as tests for poisons. The importance of this has since been seen, and more especially at a recent trial, when the judge accepted the evidence obtained in this way.

As evidence that Dr. Fagge has not confined himself to any narrow or special questions are his papers on cretinism and rickets. That on sporadic cretinism occurring in England is one of the most valuable and original which he has contributed to the science of medicine, and gave an impulse to further inquiries which have borne good fruits in an entirely novel subject. It is true that an analogous affection in adults had been described, which of late has been known as myxoedema, but Fagge showed that children at a very early age become stunted and assume the general appearance of cretins. Of these he gave full details and drawings illustrating more especially lumps or tumours at the side of the neck. Subsequently he heard of a fatal case at the Idiot Asylum, and with his accustomed energy proceeded to Redhill to examine the body. He then gave a further account of the soft fat-like tumours, which he had removed from the neck. He also mentions the fact of the absence of the thyroid body in many cases.

When it was proposed to hold some special meetings on the subject of rickets, Fagge was elected to open the discussion. He did this in a most exhaustive way, showing that he was thoroughly acquainted with all the facts and theories pertaining to the disease.

Fagge did other good work at the Pathological Society, reading an excellent paper on lardaceous disease, in which he attempted to show that it had some closer connection with syphilis than through the instrumentality of suppuration and diseased bone.

A very important communication made by him to the Royal Medical and Chirurgical Society, was that, in association with Mr. Durham, on "The Treatment of Hydatids of the Liver by Electrolysis." They published several cases, and, as showing how successful was the method, it may be mentioned that the writer occasionally sees one of the patients; she was operated on fourteen years ago, and is now the mother of a large family. Dr. Fagge also wrote a highly scientific article in these Reports on the different modes of dying, and contributed most excellent papers on "Abdominal Disease," one on "Local Peritoneal Abscess," a subject of a very obscure nature, and another on "Intestinal Obstruction." The latter contained several novel points of great practical interest, and the writer is indebted to Dr. Fagge for first fully recognising such a disease as chronic intussusception; for the prevailing notion about intussusception was that it implied acute obstruction.

Another very original and important paper was on "Acute Dilatation of the Stomach." The one on "Splenic Tumours" illustrated the great variety of forms and positions which these might assume. One ought not to forget either his treatment by saline injections in cases of diabetic coma, or his paper on phosphorus poisoning. He was not unmindful of nervous diseases, and wrote an excellent paper on "Paroxysmal Neuroses," including valuable remarks on migraine, Menière's disease, &c.

One of the latest contributions to the 'Transactions' of the Royal Medical and Chirurgical Society was a short paper on "Plumbism." It had long been known that the so-called blue line on the gums was made up of a number of dots, but Fagge took the trouble to examine the gum more thoroughly by the aid of the microscope, and he showed that the spots consisted

of rounded loops corresponding to the distribution of the blood vessels. He also showed that in some cases the intestine was similarly stained by lead: in one case the ileum was covered with spots and in another the pouch of the cæcum was quite black. Fagge showed for the first time in this country specimens of black urine in cases of melanosis; he was an active worker on the committee appointed for the investigation of croup and diphtheria.

Never was Fagge's characteristic turn of mind better exemplified than during his last illness, when he made a clinical case of himself. It was about a year and a half before his death that he became aware of the existence of serious cardiac disease, and shortly afterwards that it was probably associated with aneurysm of the aorta. When he thoroughly realised his position, he considered what course he should follow of the three which were open to him: to continue his work unheeding the catastrophe which must shortly come; to throw up everything, retire into the country, and by a life of absolute repose hope that his life might be prolonged; or to take a middle course by way of compromise, give up his hospital duties and all arduous work, see only a few patients, and during the day lie recumbent on a couch undergoing the most approved medical treatment and occupying his time by completing the book which he had had in hand for many years. This method he resolved to pursue and resolutely commenced to put it in practice. He arranged his study accordingly, put himself on a restricted diet with a limited amount of fluid, took iodide of potassium daily, and lay on his couch so that he could read and write with ease. He took accurate notes of his condition, the amount of food and fluid which he ate and drank, his secretions, his temperature, and his pulse.

His habit of case-taking seemed to require some object of interest to satisfy it even though the patient were himself. After a good trial of these means he became very hopeful and those around him gave a friendly sympathetic acquiescence to his views; but it was not very evident to them that any important change for the better had taken place. At the time of his death he was examining at the University of London, and had been occupied many hours of the last day of his life reading the candidates' papers. After retiring to his bedroom he was seized

with difficulty of breathing and in about half an hour all was over. On November 24th he was consigned to his grave in Norwood Cemetery followed by hundreds of his old pupils and friends, his coffin wreathed with flowers, the emblems of their love.

Few men at his age had done so much good work and held such important appointments as Hilton Fagge, so that the prospect before him was of the most brilliant character. In private practice, too, he was beginning to be very successful, for not only was the greatest confidence placed in him by his patients, owing to his remarkable painstaking in the investigation of their cases, but his opinion was highly valued by his confrères who sought his aid in consultation. His loss to the hospital and its school is great. A considerable sum of money has been raised to perpetuate his memory and a bronze tablet in remembrance of him is about to be placed in the museum.

The volumes on medicine which he had had in hand for some years are being edited by one of his colleagues, and when they are published, the subscribers to the 'Reports,' whilst they peruse this epitome of his life's work, will bear witness that his merits have not been overrated.

SURGICAL REMINISCENCES.

By EDWARD COCK.

RUPTURE OF THE URINARY BLADDER.

ON March 18th, 1868, I was summoned by telegram to see Mr. H—, an elderly gentleman, who resided a few miles from Colchester, and who the day previously, March 17th, at 4 p.m., had had a fall from his horse. He was a tall spare man in good health.

The accident, as described by Mr. H—, was this:—While walking gently in the road his horse stumbled and came down on its knees, bringing the rider somewhat quietly down over the horse's neck. In this relative position of the horse and rider Mr. H—received a heavy blow on the lower belly, just above the pubes, from the head of the horse, which was in the act of rising to its legs. He was able to remount, but in a few minutes felt faint and sick, and was found lying in the road and carried home. He had not passed his water since the morning, and the state of his linen and clothes evinced that none had been evacuated since the accident.

He suffered great pain in the lower belly all night, was constantly sick, and had no sleep. Early next morning, March 18th, he was seen by the late Mr. Roger Nunn, of Colchester, who passed a flexible catheter into the bladder and found it empty. We saw Mr. H— together at 4 p.m. the same day. A tumour of considerable size, well defined and circumscribed, existed between the umbilicus and pubes, just

above the latter. It appeared somewhat soft and semi-solid to the touch, and gave us the idea of a mass of extravasated blood; externally it bore every resemblance to a distended bladder; it was painful and exquisitely tender. There was a slight tenderness over the rest of the abdomen, but no positive symptom of peritonitis. He could keep nothing on his stomach, was collapsed, and very ill.

I passed a flexible catheter, and drew off nearly half a pint of healthy urine, which in the last portion contained a little blood, not recent. The emptying of the bladder caused no alteration whatever in the appearance of the tumour. I injected some water into the bladder, which returned slightly tinged. The catheter was fixed, and it was arranged that warm water should be frequently injected, and allowed to flow out through an india-rubber tube attached to the catheter. Fomentations over the entire belly. A grain of opium every three hours. Brandy as might be found necessary. He had already been placed in the most favorable position, body, shoulders and thighs raised. We saw him again the next morning, March 19th, 7.30 a.m. He had passed a quiet night, free from much pain, and had retained nourishment. I returned to town.

March 26th.—Mr. Nunn wrote to say that our patient was improving, that there had been moderate tenderness over the abdomen, but no evidence of acute peritonitis. The tumour remained the same as to size, but was less tender.

I was subsequently informed by Mr. Nunn that our patient had completely recovered from the effects of the accident, that the swelling above the pubes had gradually subsided, and altogether disappeared, but that he continued to wear an elastic catheter, having lost all power of expelling his water by natural efforts.

I visited Mr. H— December 26th of the same year (1868). He was in rude health and excellent spirits, and had gained flesh. He had passed no water normally since the accident, but had habitually worn an elastic catheter, which could be retained unchanged for four or five weeks. The urine is perfectly healthy, and there is not the slightest intolerance or vesical irritation; can retain his urine for many hours; walks and drives out, and goes to church. At the time of my visit

the catheter had been worn six weeks, and had been withdrawn nine hours. The instrument was as smooth and polished as when it had been introduced six weeks previously. He does not recognise in the ordinary way the fact of the bladder being full, but has a certain vague sensation of uneasiness after several hours' retention. The motor and sensitive nerves of the bladder are evidently paralysed.

I visited Mr. H— in 1869, 1870, and 1871. His general health remained perfectly good, and he continued to wear a catheter, which, after being retained four or five weeks, came out of his bladder looking as new as when it was put in.

Visited our old patient with Mr. Nunn December 25th, 1872. A few weeks previously he had become hemiplegic on the left side, followed by general breaking down of health and strength, and the urine is now loaded with mucus and pus, requiring the catheter to be frequently changed and the bladder washed out. He is much altered, body and mind. Mr. H— died a few weeks afterwards.

There is much room for speculation in this case; firstly, as to the nature of the swelling above the pubes which appeared soon after the accident; and secondly, as to what became of the urine which the bladder undoubtedly contained at the same time. The swelling came suddenly like blood poured out, it felt like blood, it remained as does a clot for a long time, and like blood gradually disappeared. I much doubt whether there was any connection between the supra-pubic swelling and the supposed rupture of the bladder. Mr. H— fell over the horse's neck with the belly prone, the blow was certainly very severe, and the abdominal walls were thin. Is it not probable that the extravasation of blood was produced by partial laceration of the walls? The extreme tenderness of the part precluded the attempt to make an accurate manual examination.

It was suggested that the supra-pubic tumour might have been produced by the urine from the ruptured bladder finding its way between the abdominal wall and the peritoneum lining it. The well-known and inevitable result of urine extravasated into the areolar tissue will, I think, negative this supposition. All the facts which I have related point to blood, and blood only. If this theory be correct there is still

the urine to be disposed of. There can be no doubt that there was lesion of the bladder, as proved by the blood it contained; there is no doubt that the bladder contained urine previous to the accident, and was subsequently found empty. Where could it have gone to but into the peritoneal cavity? I am inclined to adopt this explanation in spite of the fact that the symptoms of peritonitis were never of a very severe character. Possibly they were modified by the quality of the urine and its bland nature.

ANEURISM OF THE EXTERNAL ILIAC ARTERY; LIGATURE OF
COMMON ILIAC; SUBSEQUENT SUPPURATION OF SAC.

William W—, æt. 27, a working engineer, was admitted into Naaman Ward, June 14th, 1863, under Mr. Cock, who had previously seen and examined him.

History.—He seemed to have led a somewhat free and dissolute life, but was in a fair state of health. A year ago he returned from Naples, where he had been employed some time in engineering work.

Eight months ago he became aware of a swelling in the right iliac region, a little above the centre of Poupart's ligament, and at the same time was occasionally, while walking, troubled with cramp in the calf of the leg.

The swelling gradually increased in size until it assumed the formidable dimensions it presented at the time of his admission.

An aneurismal sac filled the entire cavity of the iliac fossa, and rose to the level of the umbilicus, where it inclined slightly over to the left side. It somewhat diminished in size as it rose, so as to resemble an irregular cone, the base corresponding with Poupart's ligament, and truncated apex with the umbilicus. Pulsation was distinctly felt nearly over the entire surface of the sac, and the aneurismal whirr was apparent to the naked ear and to the stethoscope. The walls of the sac did not appear to be much thickened. The right leg and thigh were much swollen, the œdema being somewhat firm, and no pulsation could be detected in the course of the femoral artery. The superficial veins of the thigh were

tortuous and large, indicating obstruction to the return of blood, but the temperature of the entire limb was fairly maintained.

The patient was put to bed, the limb slightly raised, and a bag of ice with intervening flannel applied over the sac, and its effect on the skin carefully watched. Suitable diet was ordered. At the end of two weeks some change had taken place. The size of the sac had slightly diminished, and its top was about half an inch below the level of the umbilicus, while its walls seemed to have acquired a little more hardness and firmness. The œdema of the limb had also somewhat diminished, indicating a freer return of the blood. There was little or no constitutional disturbance, and the patient was quiet and composed.

On June 30th Mr. Cock tied the common iliac artery. An incision was made about seven inches long, commencing a little above and to the inner side of the anterior and superior process of the ilium, and continued in a curved line to within a short distance of the external ring. The abdominal muscles and the fascia transversalis were successively divided until the iliac fossa was reached. Here commenced the difficulties of the operation. Blood, chiefly venous, oozed out from the whole surface of the iliac fossa, and it was constantly necessary to sponge out the wound to gain a view of the parts exposed. The peritoneum and then the sac were slowly and carefully separated by manipulation from the iliac fascia, and carried to the inner side. The upper part of the sac was thus reached, and seemed so thin that it was considered hardly expedient to detach it farther from its bed. The finger was, however, passed under the sac, and then the common iliac artery was plainly felt, pulsating freely, and apparently healthy. As far as could be judged the artery seemed to be lying at the junction of the fourth and fifth lumbar vertebræ. A little farther separation was made, and a naked aneurismal needle passed along, guided by the finger, and after some difficulty carried under the artery and brought out so that the eye of the instrument just became visible. The needle was then armed with the usual silk, and the vessel was tied without much farther trouble, but the artery itself was never fairly brought into sight. The pulsation of

the sac immediately ceased. The edges of the wound were brought together by a number of sutures, and covered by a compress of lint. Union speedily took place along the greatest part of the wound, although its entire closure was prevented by a free discharge of healthy pus. The patient's health was maintained, and in about three weeks from the time of the operation the wound was fairly healed, but the sac had undergone no diminution in size.

A new phase in the case now developed itself. The sac began to enlarge, without any pulsation, but with constitutional disturbance and fever, and it was evident that suppuration was established. In a few days the skin near the original wound became red, and fluctuation could be felt. A trochar and cannula were introduced, and brought away decomposed blood and pus. A free incision was then made, and more than a pint of pus and decomposed blood, the contents of the sac, were speedily evacuated. No untoward symptoms followed, and in a few weeks the discharge ceased, the wound closed finally, and the original sac, which had filled the iliac fossa, could scarcely be felt.

Under generous treatment health and strength were restored, the œdema of the limb gradually disappeared, and the man left the hospital in perfect health, free from any consequences resulting from the discipline he had undergone. In a short time he resumed his occupation of working engineer.

It is probable that the sac itself was not pierced by the trochar and subsequent incision, but that it had already burst, and partially emptied its contents into the tissues, which had been so freely disturbed when the artery was tied.

THE PROBABLE CAUSE OF DIFFICULTY AND UNSATISFACTORY RESULT WHICH MAY ATTEND THE ATTEMPT TO REDUCE CERTAIN DISLOCATIONS OF THE HEAD OF THE HUMERUS.

During the last sixty years I have seen a vast number of dislocations of the humerus, and in a very few cases, although the accident was of recent occurrence, the *apparent* reduction was accomplished with much difficulty and the result anything but satisfactory.

As a typical illustration of such cases I will describe one which came under my care many years ago, of which I possess some notes and which is still fresh in my memory.

A tradesman, about 50 years of age, tall and powerfully made, was sent to me from the country. He had been thrown out of his trap a week previously and had received an injury to the shoulder. Dislocation into the axilla had been diagnosed and attempts had been made by the local practitioners to replace the head of the bone, but although it could be moved and approximated towards the glenoid cavity, as soon as the force used was discontinued it resumed its abnormal position in the axilla. The great swelling of the entire arm, and the numbness and tingling pain in the thumb and fingers, indicated that the axillary vein had probably been ruptured and the brachial plexus injured. These conditions had been probably somewhat aggravated by the necessary violence used, and, as the man complained of great pain, we judged it expedient to keep him in bed for a few days with the arm raised and evaporating lotions applied. This treatment was continued for a week with considerable abatement of the swelling, and the ordinary means for reduction were now adopted. A strong man was selected to place his heel in the axilla, and a round towel adapted with a clove-hitch round the arm just above the elbow so that the strength of two or three hands might be brought to bear in bringing the arm down. At first the head of the bone obstinately refused to leave its new bed, but after great force had been used it began to move, and slowly, and unwillingly as it were, it was carried outwards until it was brought immediately under the glenoid cavity. The acromion no longer projected and the roundness of the shoulder was in great measure restored, but the unmistakable jerk and snap so grateful to the senses of the surgeon when "the shoulder is set" had been wanting and it was evident that the head was not within the synovial capsule, and that some structures were intervening between it and the glenoid cavity. The dislocation was too recent to entertain the idea that the glenoid cavity had undergone such alteration as might prevent the reception of the head.

The object now was to make the best of our failure, and retain the bone in the position to which we had brought it,

but before our arrangements could be made, the head returned to its old place as soon as the force used became relaxed. The man had suffered much and begged to be left alone for a time, and as we had cleared the road out of the axilla, thus rendering a future operation more easy, he was put to bed and lotions applied as before.

In a few days extension was again employed and without much difficulty or force the bone was brought immediately under its normal position. A pad firmly stuffed with wool was placed in the axilla and secured by plaster carried over the shoulder, while the elbow was kept in close contact with the side by a girth round the body. This was retained for a week when a strong moderate-sized globular air-pad was substituted for the wool-pad and we were glad to find that the head still retained its position with relation to the glenoid cavity.

During the next six weeks the fixings were occasionally relaxed, and fresh pads of somewhat smaller size which seemed to answer the purpose equally well and gave less pain were placed in the axilla. He then returned home, and a short time afterwards I was informed that the apparatus had been removed and that he was slowly regaining a swinging backward and forward motion of the joint, although he had not ventured to move the arm away from the body.

Two other precisely similar cases have come under my observation. In the one the attempts at reduction were abandoned as hopeless, in the other a certain amount of success was, I believe, secured.

How are we then to account for the anomalous circumstances which I have described? I believe we may explain them thus. In nearly all cases of dislocation at the shoulder-joint the capsular ligament and the tendinous expansion which partially covers it are so freely torn that in reduction the head of the bone readily re-enters the capsule through the opening by which it had passed out.

But let us suppose that the rent in the capsule is just large enough to admit of the head being forced through; the margin of the rent will then girt the neck of the bone, and when by the efforts at reduction the head is brought apparently into its normal position it is still outside the capsule, a portion

of which latter must intervene between it and the glenoid cavity.

Should we succeed in retaining the head of the humerus in its new position, and preventing its return to the axilla, the intervening portion of capsule must necessarily become absorbed before the damaged joint can be restored.

Should this explanation not be considered consistent with the facts I have adduced, I really know of no other theory which can account for the difficulty or rather impossibility of effecting a reduction in certain rare cases of dislocations at the shoulder-joint.

Since writing the above, I have found recorded in one of my note-books a fourth case of dislocation in every way resembling those I have described. It occurred under my own care at Guy's Hospital. I merely transcribe the rough record which I made at the time. "——, æt. —, a labourer, admitted August 4th, 1857, was pitched out of a cart exactly a month ago and dislocated right shoulder, was attended by a surgeon who thought he had put it right, but a week ago the head of the bone was still in the axilla. On July 30th extension by pullies was used under chloroform but without success. The dislocation was very marked. Seemed probable that the opening in the capsule by which the head of the bone had escaped was too small to admit of its return.

"Made every attempt August 4th (the day of his admission), to reduce dislocation, and found that although I could bring the head away from the axilla close under the glenoid cavity I could not bring the articular surfaces in contact, and the deformity immediately returned. Therefore adjusted the bone as nearly as possible in its normal position and kept it there by an air-pad and bandage. August the 8th the head of the bone has not in any way moved. Left the hospital a short time afterwards and is beginning under restrictions to resume the use of the arm. Did not see or hear from him again."

POST-MORTEM EXAMINATION OF THE BODY OF MR. DRUMMOND,
SECRETARY TO SIR ROBERT PEEL, WHO WAS SHOT BY
MCNAUGHTON ON JANUARY 19TH, 1843.

Mr. Drummond's back was turned to the assassin, who mistook the Secretary for the Principal, against whom he entertained some imaginary grudge.

Mr. Drummond died on January 25th, and the inspection took place the next day at the residence of the deceased, 51, Grosvenor Street. It was conducted by Mr. Edward Cock and Mr. Guthrie, junior, in the presence of Mr. Guthrie, senior, Mr. Bransby Cooper, Dr. Hume, and Mr. Jackson, senior and junior.

The surface of the body was extremely pale and exsanguineous, and there was a remarkable deficiency of hair throughout. There was a considerable layer of subcutaneous fat, more than two inches thick, on the abdomen around the umbilicus, soft, lacerable, and oily. The internal fat was abundant, and bore the same character. The countenance was mild and placid, and wore a smiling benignant expression, which fully accorded with the whole character of the man during life. The heart and great vessels contained very little blood; the clots were few and very soft. There was a small cicatrix over the ensiform cartilage, where a follicular tumour had been removed many years ago. The whole body presented an exsanguineous appearance.

There was considerable difficulty in tracing the track of the ball. It had entered the back on the left side, about two inches from the centre of the spine, passed into the pleural cavity, between the tenth and eleventh ribs, and just grazing the surface of the lung, without injury to its structure, pierced the diaphragm close to the posterior attachment of the muscle. The left pleural cavity contained in the lower part about half a pint of thin dirty-looking serum stained with blood, and in the immediate track of the ball were a few small patches of soft inflammatory deposit, loosely adherent to the base of the lung and the corresponding diaphragm. The rest of the pleural cavity was quite normal.

The ball, after piercing the diaphragm, passed through the

fat covering the renal capsule, and entered the peritoneal cavity over the upper third of the kidney. It then passed forwards through the meso-colon, and the two anterior layers of the greater omentum, perforating the latter, close to the lower margin of the stomach. It had then escaped from the abdomen by piercing the peritoneum and abdominal walls, just at the outer edge of the rectus muscle and close to the cartilage of the eighth rib, over which latter it had turned, and rested beneath the skin, on the intercostal muscle between the cartilages of the seventh and eighth ribs, whence it had been removed soon after the fatal occurrence.

The course which the bullet had taken from back to front was in great measure indicated by the ecchymosis it left in its track through the fat covering the renal capsule, and especially where the meso-colon and omentum were perforated.

There might be altogether about eight ounces of blood, or rather bloody serum, between the greater omentum and in the general peritoneal cavity, but the fluid was thin and pale, and here and there a few very soft clots might be seen. The fluid had slightly coloured the entire surface of the viscera of the abdomen and pelvis.

There had been no attempt at reparative action of the lesions which the ball had caused, and there was no evidence of peritonitis having existed. The abdominal and the thoracic viscera were healthy and normal.

A tape passed round the left side of the chest from the entrance to the exit of the bullet measured exactly thirteen inches.

It would appear that the treatment of Mr. Drummond had been throughout of a depletive character.

The fact that in the case of Mr. Drummond the ball had passed through the abdomen from back to front without injuring a single viscus or wounding a blood-vessel of importance, will probably render the account of the post-mortem examination of some interest.

McNaughton was tried for murder, and a verdict of insanity saved his life and placed him in confinement during the pleasure of the Queen.

Since writing the above I have found other somewhat similar cases of dislocation amongst my notes.

J. W—, æt. 42, admitted into Guy's Hospital, December 3rd, 1846. Nine weeks ago had been thrown from a cart and dislocated his left shoulder. The injury was overlooked. The head of the bone was deep in the axilla inwards, upwards, and forwards. He suffered no pain, and was able to move the arm very slightly. Extension was first made with the pullies directly downwards, and then with the heel of a strong man in the axilla; the head of the humerus was gradually brought directly under the glenoid cavity, but evidently had not fitted itself into the synovial capsule. Pads and bandages were applied to retain the head, but four days after he suffered so much pain that they were removed and others adopted on a better principle.

The acromion slightly projected and the position of the head was somewhat abnormally inwards and forwards. The treatment was continued, and he left the Hospital December 15th, the arm being then in a fairly good position.

January 29th, 1847.—Is beginning to regain the use of his arm, the head of the bone still a little too forward.

March 11th.—The arm has recovered its normal position and nearly perfect motion has been regained.

In this case it is possible, perhaps probable, that the bar to perfect reduction depended on the alteration which had taken place in the glenoid cavity; but it shows, among other instances, how much may be accomplished by carefully keeping the head of the bone under the glenoid cavity until it no longer is inclined to return to the axilla. I have known more than one instance in which, under similar difficulties, reduction has been abandoned as hopeless.

A. S—, æt. 27, a ship carpenter, admitted into Guy's, December 24th, 1847, under the care of Mr. Cock. His ship foundered in the Bay of Biscay, November 27th, and he then sustained several injuries, and his left arm was dislocated. No treatment had been applied. The head of the bone was deep in the axilla. Ordinary extension with the heel of a strong man in the axilla failed to move the bone. Extension

with the pulleys was then made, and the head of the bone was gradually worked outwards until it rested immediately beneath the glenoid cavity, but returned to the axilla as soon as the heel was withdrawn. The head was retained under the glenoid cavity by pad and bandages, and he left the Hospital January 16th, 1848.

There was then but slight difference perceptible between the right and left shoulder, the head of the humerus in the latter projecting a little too forwards. The ultimate result could not be obtained.

The following case has no reference to the foregoing paper, but as it is remarkable, perhaps unique, in its character, I have added it.

J. H—, æt. 20, was admitted into Guy's Hospital September 22nd, 1847. A stout, strong man. In the early part of May he had dislocated his shoulder. No treatment whatever. The head of the humerus lay high in the axilla, and had become firmly ankylosed to the coracoid process of the scapula. He could move his arm very slightly by moving the scapula; and passive motion of the arm was restricted to the extent of which the scapula was capable.

Attempts were made to break down the connexion between the two bones, but they failed.

CASE
OF
PROBABLE THROMBOSIS
OF
SUPERIOR MESENTERIC VEIN AND
RENAL VEINS.
DETACHMENT OF SEVERAL VALVULÆ CONNIVENTES
OF JEJUNUM. RECOVERY.

BY SIR WILLIAM W. GULL, BART., M.D.

THERE are points in the following case which are in my experience unique.

It was one of copious intestinal hæmorrhage, without fever, followed after some days by the evacuation of portions, more or less complete, of a dozen or more of the valvulæ conniventes (see Plate). The patient recovered, and after a period of four years is now in what appears to be good health, and at least without any further intestinal symptoms.

On the 12th of October, 1879, I was requested by my friend Mr. Venning, to see this case with him, in conjunction with Mr. Worship of Sevenoaks. The patient was a young gentleman, æt. 23, suddenly suffering from very profuse intestinal hæmorrhage. The onset of the symptoms was unexpected and began with *very severe pain in the abdomen*, which appeared to be relieved by a warm bath; and then followed the hæmorrhage. There was slight sickness, but no

hæmatemesis. The patient was apparently well up to the sudden attack of pain, eating and drinking as usual, only that some days before there had been some slight and transient œdema of the right hand and part of the forearm, which could not well be accounted for, and which had disappeared at the time of the attack. The œdema was vaguely though probably truly referred to a secondary taint, a primary sore having been contracted five months before; but secondary spots on the skin were doubtful.

On passing the intestinal evacuation of blood from one vessel to another, it was noticeable that it varied in colour, from the darkest venous with black coagula, to that of a lighter tint, indicating that the bleeding surface was extensive and high up in the intestine. The warm bath appeared to have given great relief, and the patient expressed himself as feeling as if something had been set free by it, but this relief was probably due to the hæmorrhage.

The question which naturally engaged the attention of my colleagues and myself was, as to the pathological cause. There had been no fever, no previous illness, and nothing complained of, but the slight transient œdema mentioned as occurring a few days before. The conclusion was, that the cause was mechanical, and one that obstructed the mesenteric vein high up. The severe pain, the bleeding, and the character of the blood, favored this opinion as to the seat of the obstruction. It was conjectured, also, that the obstructing cause was outside the vessel, and might be a gummatous deposit. Whether it were so or not, cannot be settled, though the subsequent history supported this opinion. Assuming it to be so, it was to be expected that other veins in the neighbourhood, as the renal veins, would suffer; and this subsequently occurred. The urine at the time of the attack was carefully examined. It was pale, sp. gr. 1015, slightly opalescent by heat and nitric acid.

On the night of the 15th, three days after the attack, the patient fell into a state of sudden collapse, and became almost pulseless. The following day the temperature was 100.8° , pulse 108, abdomen tympanitic, constant vomiting, ejecta bilious without blood, great thirst. On the 17th, symptoms less urgent, urine with even less evidence of albumen, temperature

99.4°, pulse 90. On the 18th, pulse 84, no sickness, evacuations bilious and without blood; the two previous days they had been melænous. Urine darker in colour, sp. gr. 1020, a large amount of albumen. On the 20th and 21st, sloughs of *valvulæ conniventes* were evacuated, but with no trace of muscle-tissue attached to them. From this day to the 27th, portions of mucous membrane, rings or portions of rings (*valvulæ conniventes*), were passed in the evacuations, the free surfaces being rich in villi. On the 27th, the last of these sloughs came away. The patient was going on well, abdomen soft, evacuations formed and natural in appearance. There were no external swellings, or nodes, or oedema in any part of the body. The renal secretion, however, continued to undergo remarkable changes, though the patient was otherwise convalescent. On Nov. 7th, urine acid, of a dull amber-greenish colour, sp. gr. 1025, one quarter to one half its volume of albumen deposited on boiling; casts with hæmatin, and exudation cells; pure hyalin casts; small separate concretions of crystalline hæmatin.

Nov. 21st.—Urine acid, depositing urates and a peculiar clot of loose translucent bloody mucus, loaded with albumen, sp. gr. 1031, after separating albumen by boiling, sp. gr. 1025. There had been no further intestinal symptoms since the 27th of October.

On the 17th of January of the year following (1880) the same abnormal state of the urine continued: sp. gr. normal, much albumen, and still a deposit of the same peculiar bloody mucus. This was suspected to come from the pelvis of the right (?) kidney. Notwithstanding this condition of the urine, the patient was convalescent, appeared to be in his usual health, and has so continued up to the present time, November, 1883. He has been able to travel and otherwise enjoy himself in his usual manner like a healthy person, but the urine continues to be albuminous, though of normal sp. gr., and free from the loose translucent bloody mucus, which occurred three years ago.

In the sudden onset of pain, the case accords with what is known of sudden distension of vessels, whether from thrombosis or dissecting aneurism. Clinically, this fact of sudden and severe pain in the abdomen due to sudden distension of

vessels, has to be borne in mind in diagnosis, so as to separate the cases from attacks of ordinary colic, the pain of gall-stones and of renal calculus. As the patient whose case is here given recovered, and is now apparently in health, the pathological history is happily defective, but it may in part be supplemented by what occurred in a case recorded by the late Dr. Hilton Fagge.¹

A lady, æt. 34, of pale and sallow complexion, and having a slight scar in the lumbar region, from an abscess (?) in childhood, had given birth to a healthy child a month previously; and was suddenly seized, at 6 a.m., with violent pain in the abdomen and vomiting of a rather viscid blood-stained fluid; symptoms of collapse; abdomen flaccid; no tenderness anywhere, nor unnatural fulness. "I could not," says Dr. Fagge, "form the slightest idea as to the cause of this sudden illness. Patient died at five o'clock p.m. eleven hours from the beginning of the attack."

A post-mortem examination was held the following day. "When the abdomen was opened the first thing that attracted observation was extreme congestion of part of the small intestine. The congestion began about four inches from the termination of the duodenum, and ended at about the middle of the small intestine. The internal surface of the bowel was deeply reddened, and covered with shreds of mucus, so that it seemed as though the mucous membrane itself was being detached. The congested state of the jejunum was so like what would have resulted from internal strangulation of the bowel that I searched very carefully for any hernia or other cause of obstruction. But none could be found. Moreover, the affected part of the intestine was by no means distended; it was not larger than the rest. The real explanation of the state of the intestine, however, became apparent when we cut through the mesentery to remove it. The branches of vein coming from the affected part were found to be distended with adherent coagulum. This condition was traced up the superior mesenteric vein into the trunk of the vena portæ. The rest of the abdominal viscera were apparently healthy. The vena cava had by no means been in a normal condition before the thrombosis began. It was flattened and narrowed,

¹ 'Pathological Transactions,' vol. xxvii, 1876, p. 124.

and embedded in a quantity of very firm fibrous tissue. This induration extended downwards along the brim of the pelvis and down the sides of the lumbar vertebræ. The intervertebral discs were all healthy, and the interior of the bodies of the vertebræ appeared perfectly normal. But the left side of the last lumbar vertebra seemed to be slightly excavated, and on scraping it with the knife I found that it felt rather rough, and that I could cut away some portions of osseous substance embedded in the tough fibrous tissue.”¹

This case of Dr. Fagge’s probably supplies the evidence wanting in that I have here recorded. The evidence of an old affection of the connective tissue around the vessels in the abdomen, the pale and sallow complexion of the patient, and the old scar in the lumbar region, show an old-standing cachexia; and there being no mention of tubercle, this cachexia may probably have been due to an inherited taint of syphilis. If so it would further accord with the history of the case given above, in which there was a plain history of taint, though no external evidence of tertiary effects. Without laying undue weight as to the parallelism of the two cases as respects their latent pathology, it will be admitted to be a question to be raised in similar instances how far a syphilitic cachexia, affecting the connective tissue (gummata) around vessels, and causing hæmorrhage, is not to be placed side by side with disease of the connective tissue around nerve trunks from the same cause. In the case here given my colleagues and myself agreed to treat the case as one of this kind, and prescribed the iodide of potassium and bichloride of mercury, notwithstanding the highly albuminous state of the urine; and the least that can be said is that the patient is well, except for what may possibly be referred to one (?) of the kidneys. It further seemed to us probable that only one

¹ Dr. Frederick Taylor has also brought before the Pathological Society (*‘Transactions,’* vol. xxxii, 1881, p. 61) a case of a somewhat similar kind. A child, æt. 5, was suddenly seized one afternoon with abdominal pain and vomiting; she had violent paroxysms of pain all night, and died the following morning. On post-mortem examination blood was found effused into the peritoneal cavity, and into the cavity of the small intestine. The jejunum and ileum were deep red in colour. The superior mesenteric artery was found obstructed by being matted together with a fibrous mass in the mesentery; and the mesenteric vein contained a thrombus at the same point.

kidney had suffered from the venous obstruction, and this one severely, as shown by the state of the urine, and especially by the deposit in it of the peculiar loose, bloody, translucent mucus, which has been mentioned, and which we attributed, rightly or wrongly, to changes in the renal pelvis. The affection of one kidney alone is suggested by the course of the case, and this would be compatible with the patient's good health, although the renal secretion has never recovered its normal state.

In respect of the sloughed *valvulæ conniventes* it is to be remarked that the sloughs differed entirely from what would have been passed in intussusception; they were unaccompanied by any trace of muscular coat. What really occurred is probably explained by the post mortem in Dr. Fagge's case. He states that at the affected part it seemed "*as though the mucous membrane itself was being detached;*" and this occurred in the case here recorded, as shown by the detached *valvulæ conniventes* thrown off in broken portions. This case suggests some thoughts as to therapeutics: the uselessness, if not harmfulness, of trusting symptoms. The profuse hæmorrhage would have seemed to call for styptics, yet the hæmorrhage was useful and curative, and drugs to stop it would have done nothing but harm. The hæmorrhage was a necessity of the conditions, and therapeutical. On the question of artificial depletion it supplies some lessons. Bleeding did good in this case at least.

I am indebted to Dr. Charles Hood for separating the sloughs of mucous membrane from the fluid in which they were passed, and also for arranging them as seen in the drawing.

Report by Dr. Goodhart.—The specimen as preserved in the bottle and extended upon talc is ten and a half inches long, but there are many gaps in this length to reduce its actual measurement. It consists of alternating thin and thick bands of membrane, the latter corresponding no doubt to the *valvulæ conniventes* of the intestine, and the thin part to the mucous membrane between them. Those parts which are thicker, but all more or less, have the velvety appearance characteristic of the villous surface of the intestine. Some are strips of membrane representing half the circumference of the small

intestine, others more, and many are complete rings of the entire circumference of the bowel.

No parallel bands of muscular fibre can be detected on the closest examination and by transmitted light.

There can be no doubt that a localised enteritis and necrosis of the mucous membrane has occurred, and that the mucous membrane has separated in consequence. But it is remarkable that the disease should have been at the same time so severe, and yet so superficial that the muscular coat is entirely spared.

DESCRIPTION OF PLATE.

Coils of valvulæ conniventes passed in the evacuations, the inner surface velvety from villi.

得得

得得

ON A CASE
OF
FRACTURE OF THE SKULL,
IN WHICH
CEREBRO-SPINAL FLUID ESCAPED FROM THE VAULT
FOURTEEN DAYS AFTER TREPHINING.

BY R. CLEMENT LUCAS, B.S.

IN the 'Guy's Hospital Reports' for 1876, 1878, and 1881, will be found the records of two very interesting and exceptional cases of simple fractures of the vault of the skull, which were followed by tumours of cerebro-spinal fluid beneath the scalp. These cases I was fortunate in keeping for a long time under observation. One died of acute meningitis a year and nine months after the injury, and the post-mortem examination is given in the 'Reports' for 1878. It showed that the original injury had reached the ventricle. The second case is related in the volume of the 'Reports' for 1881 and the report is there carried up to two years and seven months after the injury, when a pulsating gap was still to be felt in the forehead along the line of fracture.

From the study of these and other cases I drew two conclusions, viz :

1. That cases of simple fracture of the skull followed by collections of cerebro-spinal fluid beneath the scalp are peculiar to children.

2. That when cerebro-spinal fluid escapes through the vault (whether the fracture be simple or compound) the injury has extended to the ventricular cavity.

The following case is one which serves to support the correctness of the second of these two inductions; and had the symptoms been less urgent, so that it might have been justifiable to postpone operating, it is highly probable that a tumour of cerebro-spinal fluid would have formed beneath the scalp as in those cases already reported. I make this statement because all the conditions were present which in other instances have determined this rare complication of fracture of the vault. The child had fallen from the landing of a staircase and dropped a distance of about twelve feet on its head. It was but a year old, an age at which the thinness of the bones and the yielding nature of the skull allow of extensive damage to the brain, without any laceration of the scalp. Further, the post-mortem examination revealed that laceration of the brain substance had reached the ventricular cavity, whilst rupture of the dura mater was noticed at the time of the operation. The escape of cerebro-spinal fluid from the wound on the fourteenth day corresponds closely with the time that has been noticed in other cases, and probably indicates that subsequent to the laceration a softening process supervenes, together with an increased secretion of cerebro-spinal fluid from inflammation, so that pressure from within and yielding of the brain wall together contribute to the escape of fluid from the ventricles.

Simple depressed fracture of the skull. Trephining followed by relief of symptoms. Escape of cerebro-spinal fluid noticed on the fourteenth day. Erysipelas on the twenty-first day, followed by meningitis and death.—The following report was made by Mr. E. Stanley Tresidder; T. C. S. H—, æt. one year, was admitted into Guy's Hospital on June 18th, 1881. The parents were somewhat delicate, both having suffered from enlarged glands and abscesses in the neck. The child was born with a congenital hernia, and had suffered from bronchitis.

At five p.m. on June 18th the child fell down the staircase, a distance of twelve feet, and pitched upon its head. It was not unconscious when picked up, but soon afterwards vomited. The child was admitted about an hour afterwards. Before

admission it had a clonic convulsion of the right side affecting the right arm, leg, and right side of the face, lasting about half an hour.

About an hour after admission it had another clonic spasm of the right side, which also lasted about half an hour.

There was a swelling extending over the left side of the head, and above this, two inches from the sagittal suture, was a distinct depression running longitudinally through the frontal and parietal bones. The child was very white and was noticed to be paralysed on the right side. The pupils were both dilated, the left more than the right. Mr. Lucas was called to see the case five hours after its admission and determined to trephine at once.

The fracture extended from the frontal bone some distance backwards through the parietal, but how far was not ascertained. Chloroform having been administered, a crucial incision was made over the seat of fracture, and the periosteum and scalp were raised together. The bone being removed, the dura mater was seen to be torn through in the line of the fracture, and the brain substance was exposed. A thin blood-clot was seen situated on the dura mater, and was not removed. The operation was performed under carbolic spray, and carbolised gauze dressings were employed. An ice bag was afterwards applied to the head.

June 19th.—It was noticed that all paralysis had disappeared.

20th.—Temperature in the morning 100°. The child is somewhat fretful, but takes the breast well, and slept better last night. There are no signs of paralysis, arms and legs moving freely. The bowels have not acted. The pupils are normal. The head was dressed under the spray.

21st.—Temp. 98·4°. Still fretful, but takes the breast well. Pupils normal. The bowels have not acted. It was ordered Pulv. Rhei co. gr. iv, Hydrarg. cum Cretâ gr. j, statim.

22nd.—The child seems much better to-day. He is hungry, and the mother says the breast does not satisfy him. The tongue is clean and moist, and the lips not dry. The bowels have acted twice. The wound has been dressed. Temp. 98°.

23rd.—The child has been dressed to-day, and the four wire sutures removed. The wound appeared to be united throughout by primary union. Temp. 97.6° .

24th.—The child continues well. His bowels have acted to-day. The wound was dressed. Temp. 98° .

27th.—The temperature has continued normal or a little below. The ice bag has been discontinued.

28th.—The child was allowed bread and milk.

29th.—The mother does not notice any difference in the condition of the child, and thinks it as well as before the accident.

July 2nd.—When dressed it was noticed that, if the child cried or moved a little, clear fluid trickled out of a minute aperture where the incisions cross. Antiseptics have been left off, and dry lint is now applied.

6th.—The registrar made the following note:—There is a small sinus from which, when the child cries, a drop of clear fluid exudes. It resembles cerebro-spinal fluid. The dressings are not soaked when removed.

9th.—The child continued quite well until to-day, and it had been arranged that it should leave the hospital to-morrow, as the mother wished to go home. At midday it became restless and afterwards drowsy, in which condition it continued during the remainder of the day. At the same time a pink blush was noticed over the forehead and behind the ear, and in the evening the scalp was swollen. At 9.30 p.m. the wound was dressed, and a little pus oozed out.

10th.—The child became unconscious this morning. The temperature ran up to 102° in the morning, and in the evening reached 103.4° . Redness this morning has nearly disappeared, and the swelling also, except behind the ear. The child is very pale, but continues to take the breast. It cries out occasionally.

11th.—The child lies unconscious, with the eyes fixed and glassy. Pupils contracted, with slight ptosis on the right side. Skin feels cold to touch, but in the axilla the thermometer records 101.4° . The left corner of the mouth and left sterno-mastoid work convulsively. Left arm swings in a circle, and tears at the wound. Respiration laboured, 48 per minute. The wound and surrounding skin have sunk very

perceptibly. Pulsation can be seen in the depression. It died in the course of the morning.

A *post-mortem examination* was made on the same day by Dr. Goodhart.

There was an extensive fracture running from the trephine wound on the left side of the skull forward well into the frontal bone, and a rectangular continuation behind running upwards towards the sagittal suture. There was much blood extravasation between the skull and the scalp. There was a little purulent lymph lining the side of the skull and the middle fossa, but ceasing above before the fracture was reached, and there was about half an ounce of green pus in the middle fossa on this side. As this could not be traced to the trephine hole or fracture Dr. Goodhart thought it might have come there by gravitation, and not by direct extension of the inflammation. There was no fracture of the base, and no pus in the tympanum. The other viscera were healthy. The crucial wound for trephining had healed except a small central aperture, which had discharged clear fluid, thought to be cerebro-spinal.

The calvaria and brain were removed together, and placed in spirit to harden before being subjected to further examination.

When the hardening had been completed, a section was carefully made transversely through the trephine wound, skull, and brain, and this showed very clearly that the brain had been damaged as far as the lateral ventricle; and, further, that there existed a track along which cerebro-spinal fluid had passed from the ventricle to the surface of the scalp. One half of this section has been presented to the museum of the Royal College of Surgeons, and the other half, from which the engraving has been made, is deposited in the hospital museum.

Remarks.—Although this case terminated fatally, the child's death was due to one of those unfortunate accidents which often rob surgeons of their successes, when these appear almost complete, and when precautions seem no longer necessary. No surgeon would, I think, have postponed operating with such symptoms present as have been narrated. After the fall, the child had had two right-sided convulsions, each

lasting it was said about half an hour, followed by right-sided hemiplegia. There was a depressed fracture on the left side, and the left pupil was more dilated than the right. The symptoms indicated both irritation and pressure, and were relieved in a remarkable manner by trephining and elevation of the bone. It was noticed after the bone was removed that the dura mater was torn and the brain exposed. Still, in spite of the severe intracranial laceration and the extravasation of blood both beneath the scalp and between the dura mater and bone (the latter of slight extent), the wound healed primarily and it was for some time thought completely. The temperature on the second day after the operation was 100°, but after this it fell to normal and remained so for nearly three weeks, during which time the child appeared quite well, intelligent, and free from paralysis or convulsion. In spite of the extensive damage to the brain which the necropsy revealed the child was never sick; it took the mother's breast with avidity, and soon appeared desirous of more substantial diet.

On June 28th, ten days after the injury, it was allowed bread and milk, as the mother's milk no longer satisfied it. The day following, the mother thought the child quite as well as before the accident. The antiseptic dressings were now dispensed with as it was thought the wound was healed, and a pad of dry lint was placed over the site of operation.

On July 2nd, fourteen days after the injury, a little clear fluid was noticed to escape from a minute aperture at the centre of the crucial incision; still, there were no indications of cerebral inflammation, the child continuing in its usual health. Small quantities of this fluid, which could be nothing but cerebro-spinal, were then noticed to escape when the child cried. The mother had become very anxious to return with the child to her family, and it was only the occurrence of this symptom which caused us to delay her departure from the hospital. We watched the case for five or six days longer, and then, as the child continued apparently quite well, in spite of the occasional escape of cerebro-spinal fluid, it was agreed that the mother should leave on July 10th. On the 9th the child became restless and irritable, crying and refusing its food, and later it became drowsy.

At the same time a red blush was noticed over the forehead and behind the ear, and it was evident it had taken erysipelas. As a patient had been removed from a neighbouring bed on account of an attack of erysipelas a day or two before, there cannot, I think, be any doubt as to the nature of the inflammatory attack. On the following day the child became unconscious, and its temperature gradually ran up, reaching 103.4° by the evening. On the third day of the fever the case terminated fatally.

The communication of the wound with the ventricular cavity of the brain, which was demonstrated by post-mortem examination, is the chief feature of interest in the case, and the principal reason for its publication. I showed in my last communication that there was no post-mortem evidence that a wound of the visceral layer of the arachnoid was sufficient to account for the escape of cerebro-spinal fluid from the vertex. Such teaching rests only upon conjecture, and is, I believe, erroneous. Were it true, the appearance of cerebro-spinal fluid ought to be much more frequent than it is in cases of compound fracture of the vault. For it is certain that in many instances in which the dura mater and brain are known to have been torn no escape of clear fluid has occurred. On the other hand, in every case in which an autopsy has taken place, the communication between the wound and the ventricular cavity has been demonstrated. Nor is the fact, that several cases in which this symptom has been noticed recovered, any argument against the probability that the brain has been deeply penetrated. It is astonishing to witness to what an extent the hemispheres may be lacerated without occasioning death or even very urgent symptoms. Children, indeed, whose intellectual faculties have not been developed, as soon as the pressure on the reflex ganglia is relieved, suffer little from the most extensive cerebral lesions, provided meningitis does not ensue. Of the three cases I have now put on record in these 'Reports,' all recovered from the immediate effects of the injury, and it was the addition of a second cause which placed a post-mortem examination within my reach in two of the cases. The first, though subjected to the most gross neglect, resulting in severe rickets, lived a year and nine months and then died of acute meningitis. The second is

still living and well, and the third succumbed to an attack of erysipelas at a time when it was believed to be out of danger.

Further report, five years and a half after the accident, of R. P—, a case of Fractured Skull followed by a collection of Cerebro-spinal Fluid beneath the Scalp. (Published in the 'Guy's Hospital Reports,' 1881.) By R. CLEMENT LUCAS, B.S.

The boy, now aged seven and a half, when a year and eleven months old fell from a window a distance of ten feet and fractured his skull. There was no wound, but the line of fracture could be felt distinctly, running from the left orbit obliquely across the forehead towards the anterior fontanelle. Fifteen days after the accident the swelling (at first due to blood which had been gradually decreasing) suddenly increased rapidly in size. It was noticed to pulsate and to become especially prominent when the child cried. The child recovered, and the report is carried up to two years and seven months after the accident. At this time the fissure was still wide, at one part a quarter of an inch in width, and apparently closed by fibrous tissue.

He was brought to see me on February 4th, 1884. Some fulness is still to be seen on the left side of the forehead, which varies, it is stated, at different times, especially after a fit of crying. The mother also states that he complains of pain in the forehead, and there appears to be an increased fulness when he is masticating a hard crust of bread. The child attends the Board School, and is said to be intelligent. The line of fracture is still to be clearly felt running obliquely across the forehead, but the gap is much narrower, and there is no pulsation to be felt in it.

DESCRIPTION OF PLATE,

Illustrating Mr. Lucas's Case of Fracture of the Skull.

FIG. 1.—A. Transverse vertical section through the left hemisphere of the brain, opposite the trephine hole.

B. Section of longitudinal sinus.

C. Falx cerebri.

D. Ventricle of brain.

E. Tract through the brain by which the fluid from the lateral ventricle escaped at the trephine hole.

F. Portion of scalp covering trephine hole.

G. G. Bone on either side.

H. Dura mater.

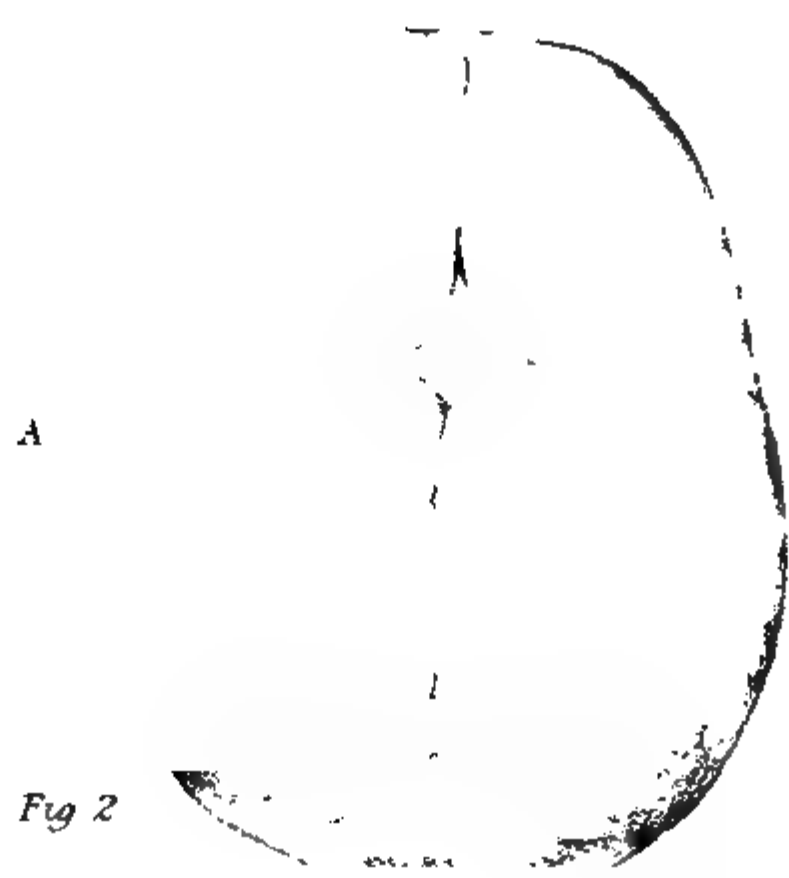
FIG. 2.—Diagram of the skull indicating the line of fracture and the position of the trephine hole.

A. Trephine hole.

A

C

D



GANGRENE
OF THE
SKIN AND CELLULAR TISSUE OF THE ARM
FOLLOWING VACCINATION.

By R. CLEMENT LUCAS, B.S.

E. G. P—, æt. 5 months, was brought to see me at the Evelina Hospital for Sick Children on Wednesday, February 28th, 1883, on account of a gangrenous condition of the skin of the arm following vaccination.

The mother was a delicate-looking woman; the father a strong, tall, well-built man. The parents had been married six years, the father's age being twenty-seven. There had been three children as the result of the marriage, none of whom had been suckled by the mother.

The first child was born thirteen months after marriage. This child had a rash over its buttocks when six weeks old and a thrush in its mouth, but the mother was doubtful as to snuffles. It had no medical treatment, and got well when Fuller's earth was used, and the buttocks were washed with oatmeal and water. The mother never suffered from an eruption of any kind, either during her pregnancy or after the birth of this child. This child was brought up on the bottle, and had rickets when a year old, but is now strong and well.

The second child was born about two years ago. It likewise had a rash over its buttocks when about six weeks old, and the mother thinks it also had snuffles. It recovered

without medical treatment, and, though bottle-fed, escaped from rickets. The mother has never had a miscarriage.

The third child, who is the patient, was born on the 10th of August, 1882. It had snuffles at birth, and a rash over its buttocks when three weeks old. It also had thrush in the mouth. It was treated by a medical man, who gave it grey powders, &c., and the rash disappeared. It has been fed on condensed milk, Ridge's food, and Robb's biscuits. It was thin and weak when taken to be vaccinated. When three months old it was taken to the Surrey Chapel station, Blackfriars, to be vaccinated. The first time it was vaccinated it did not "take"; accordingly, a week later, it was vaccinated in the same place again, but again the vaccine failed to produce vesicles.

The child was not taken again until it was five months old. It was then vaccinated for the third time in the same site, and the following week five vesicles had developed. No child was vaccinated from this infant, nor was any lymph taken from its arm. Between the second and third vaccination the mother had noticed that the child had grown thinner. The last operation was performed in January, 1883, and about three weeks later the skin at the site of the operation turned brown and sloughed. She took the child to a private medical man, who treated it for a short time and then advised her to take it to the Evelina Hospital.

I saw the child first on February 28th (it had been previously seen by the house surgeon in my absence), when it presented the following appearance:—It was extremely emaciated, with sunken cheeks and eyes, and wasted limbs. The abdomen was tympanitic, and there was no enlargement of either liver or spleen. There was no cranio-tabes or enlargement of any epiphysis, and the ribs were not beaded. The buttocks and pudendum were in a state of intertriginous eczema, but there were no disseminated shiny spots, and no eruptions on any other part of the body. The mucous membrane of the mouth and lips was sound.

The left arm was slightly swollen, and at its upper part presented a somewhat remarkable appearance. A sore commencing at the point of the shoulder extended down below the middle of the arm, and was occupied in the centre by a

large, thick, black slough. The sore was two inches and one eighth in length by an inch and a half in breadth. It presented a sharply defined edge of ulceration, which dipped through the skin into the cellular tissue beneath, and a red blush of injected vessels extended for about half an inch around. Between the slough and ulcerating edge was a yellow line coated with pus. The slough, which was hard, black, and dry, was divided into two portions, the upper of which was oblong in shape, an inch and a half in vertical measurement, one inch across, and a quarter of an inch in thickness. The smaller portion of slough was situated below and in front of that already described, and was about three quarters of an inch in diameter. There was no glandular enlargement in the axilla.

The child was ordered cod-liver oil and steel wine, and carbolic oil was applied to the wound.

It died on March 4th, having gradually sunk without any convulsion or special symptom of note.

The father of the child was seen after the child's death and questioned as to syphilis. He most emphatically denied ever having had a venereal disorder of any kind, either before or after marriage.

A post-mortem examination was obtained on March 6th, in making which I was assisted by Dr. Newsholme, the registrar, and Mr. Newnham, the house surgeon of the Evelina Hospital.

The head was not examined. The heart was healthy. The lungs were collapsed at the lower part behind. The intestines were empty, and stomach small. The liver was healthy and the spleen also, but contracted. The kidneys were also healthy.

On inquiring of the surgeon who vaccinated the child if anything had been noticed in other cases operated upon at the same time, I was informed that four other children had been vaccinated from the same source, and that no ill result had followed in any of these cases.

The following is an extract from the letter of the Public Vaccinator: "The child of which you write was vaccinated from the same source as four others, all of which proved most successful, and without any complication afterwards being brought to notice."

Remarks.—Among the multitudes who are subjected in the course of every year to the influence of vaccination accidents are so infrequent, that we might almost regard the operation as removed from danger. But such immunity ought to make us all the more careful to trace, if possible, any apparent misfortune to its true source. Nor do I think that the ill-judged prosecutions and tirades by agitators should make us less ready to discuss the cause of any accident that may occur to a vaccinated infant. These rare cases, when examined and carefully sifted, will almost certainly show that some other influence than the introduction of pure lymph was the true cause of the disaster.

From the history of the foregoing case it will be noticed that the infant had difficulties other than vaccination to contend with, and it is almost certain that the child's general low state of health had more to do with the character the wound assumed than the operation had to do with its collapse.

The first question which will suggest itself to the mind of any reader is, whether syphilis might have been the cause of the gangrene at the site of the vaccination, and if so, whether it was introduced with the lymph or whether it previously existed. That it was not a case of vaccine syphilis I think we may decide with absolute certainty, since the records at the vaccination station show that four other children were vaccinated from the same source, and in these no untoward circumstance occurred. Moreover the wound did not present the characters of hard chancre, and—what I think of even greater importance—there was no glandular enlargement in the axilla or elsewhere. The child had an eczematous eruption about the buttocks, but this had made its appearance some months before the vaccination, and could not therefore be attributed to any poison introduced at that time.

Beyond the suspicion which the red rash on the buttocks might suggest, there was absolutely no sign about the child which could be taken as indicating active syphilis, the mucous membranes and skin in other parts being at the time of examination free from eruption. Putting on one side the chance of vaccination syphilis, there remains to be discussed the question of hereditary disease as possibly determining the character of the sore. The tendency of sores in late syphilis to assume a

phagedenic character is generally admitted, and the occurrence of gangrene in a person whose constitution has been shattered by syphilis may perhaps be attributable to the influence of that disease. I searched very carefully into the history of the case to discover any such taint, and the family history, as indicated by the occurrence of rashes on other children, is sufficient to excite ground for suspicion. The mother, however, denied all knowledge of infection, and also that she had suffered from any skin affection or sorethroat; but this, even if honestly given, does not disclaim for her children the presence of taint, since it is well known that the children may give evidence of the disease when in the mother it is completely masked.

The denial on the part of the mother is of little moment as a matter of evidence, since apart from the natural bent in woman to defend her modesty at the expense of her veracity, there remains the fact—as pointed out by Colles—that she may become infected through her child without having herself any secondary symptoms. The evidence of a father is generally more reliable, but not to be accepted altogether without scrutiny. In this case the father would allow no doubt to rest upon his morality, and denied absolutely that he had ever suffered from a venereal disorder. It may be argued in support of the man's statement that he was married at the early age of twenty-one, and thus his morality was put to a shorter test than usual; whilst supposing it to have failed, he was exposed for a shorter time to the risk of infection. Leaving the history of the parents and searching among the children for evidence of the disease we discover the somewhat remarkable and suspicious fact that all the children suffered at an early age from eruptions on the buttocks. The eldest child was born thirteen months after marriage and had an eruption at the age of six weeks, which, however, seems to have disappeared without any special treatment. I could not obtain any definite history of snuffles in this child. The second child seems to have had both rash and snuffles, but to have recovered like the first without requiring any special medical treatment. The third child was the patient whose history is detailed and there seems no doubt that it had both snuffles and a rash on the buttocks. At the time, however, it was brought under my notice, the eruption on the buttocks

presented none of the characters which one might consider characteristic of syphilis. It was a uniform eczema confined to the parts wetted by the excretions, whilst the mucous membranes were clear. The case would appear to have been treated, when the child was about a month old, with grey powders, and to have been relieved, but it is so much a routine practice to treat infantile eruptions in this way, that I doubt if much reliance is to be placed upon this circumstance as an aid to diagnosis.

Taking the evidence as a whole I am inclined to doubt the existence of syphilis, and to attribute the rashes to the artificial diet and neglect of cleanliness. The inability of the mother to suckle her children and the consequent injudicious feeding to which the children were subjected is, I think, alone sufficient to account for the imperfect health suffered by these infants. Those who are connected with children's hospitals in London know only too well how great a bulk of out-patient practice is contributed by these two factors, innutritious diet and lack of cleanliness. Irregular bowels, at one time constipated, at another subject to violent diarrhoea; prolapse of the rectum; strophulus, eczema and impetigo; ulceration of the nostrils, tinea tarsi, and phlyctenular ophthalmia; cuticular and cellular abscesses with secondary inflammation of the glands; relaxed ligaments, distorted bones and general rickets; these are the cases which crowd the out-patient rooms and are dependent mainly upon the conditions named. To the deleterious influence of these two factors the vaccinated infant was subjected, and twice, probably owing to the low state of its health, the lymph failed to excite the ordinary pustules. Meanwhile its general health appears gradually to have declined and when it was vaccinated for the third time the inflammatory action which succeeded became of a virulent and gangrenous character. The post-mortem examination failed to show any internal evidence of syphilis or indeed of any disease; but the intestines were empty, the solid viscera small and pale, and there was an absence of fat, as one would expect in a case of prolonged starvation. The conclusion to which I came, after careful consideration of the case, was that the gangrene at the site of vaccination was secondary, and dependent upon the low general state of the child's health.

Only one similar case has ever been brought under my notice, and this was shown me some weeks before by my colleague Dr. Frederick Taylor. In his case, as in the one I have related, there was raised a question of syphilis, and it was in reference to this that my opinion was asked. There was the same black patch, and also if my memory serves me aright, a similar eczema about the nates with extreme emaciation, but no glandular enlargement, nor any general evidence of syphilis.

DESCRIPTION OF PLATE.

Fig. 1 illustrates Mr. Lucas's Case of Gangrene following Vaccination. (Page 31.)

- A and B. Dry gangrenous portions of skin and cellular tissue.**
- c. Surface covered with pus between the slough and living skin.**
- D. Ulcerated and inflamed margin of living skin.**

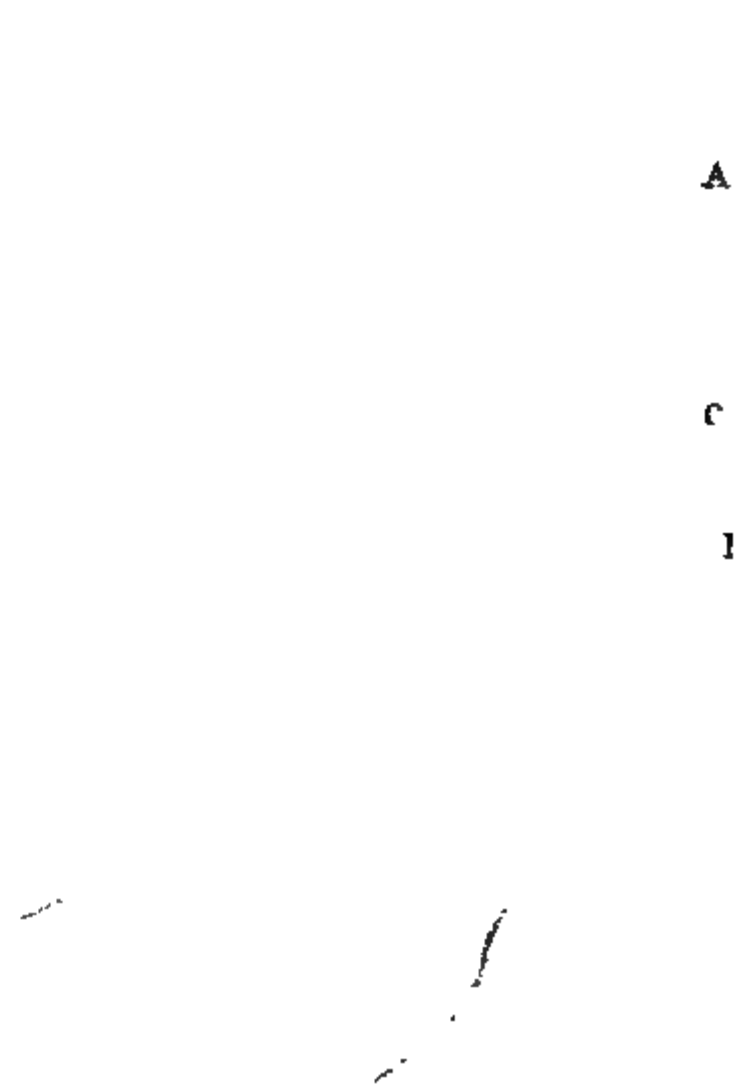
Fig. 2 illustrates Mr. Lucas's Case of Unusual Form of Colles's Fracture. (Page 375.)

- A. Anterior surface of lower fragment.**
- B. Carpal articular surface directed downwards and backwards.**

Fig II



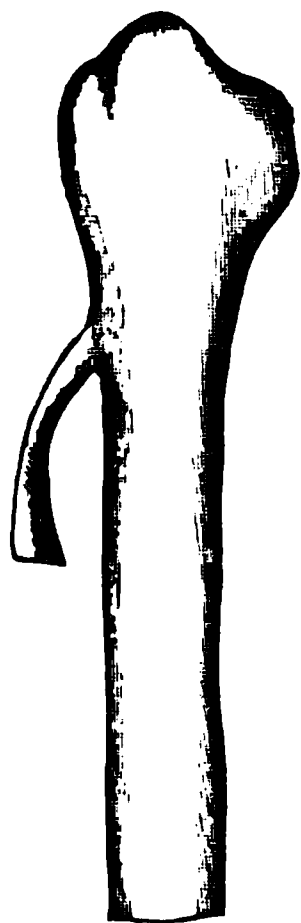
Fig. I



AN ACCOUNT
OF THE
ABNORMALITIES OBSERVED IN THE
DISSECTING ROOM
DURING THE WINTER SESSION 1882—1883.

By P. HOBBOCKS, M.D., W. HALE WHITE, M.D.,
AND
W. A. LANE, M.S.

WE should not have thought it worth while to write a paper, when the abnormalities were so few, had it not been that we wished if possible to make the recording of this dissecting-room paper an annual feature of the hospital reports. Unfortunately, during the last winter session, we have had somewhat fewer subjects, and when it is remembered that a large number of abnormalities are not recorded because students do not call the demonstrator's attention to them, the brevity of this paper admits of an easy explanation. Then again, in many cases, very common abnormalities, so common in fact that they might almost be regarded as normal—such, for example, as the posterior scapular coming from the third part of the subclavian—have not been recorded, as they are mentioned in all the better text-books, and have been noted in previous volumes of these reports.

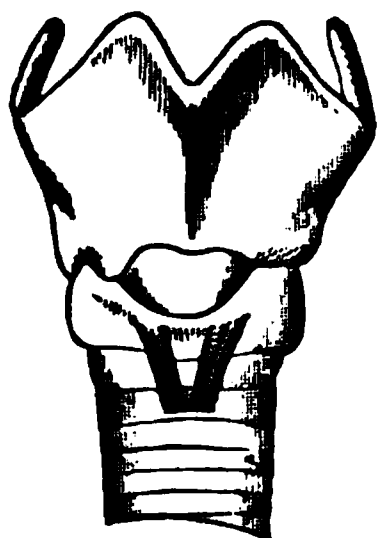
BONES.**FIG. 1.**

The only abnormality we have to record is an abnormal process from the fibula. It occurred in a male subject on the right side only, and consisted of a process of bone about an inch long, which sprung from the internal surface of the fibula in front of the oblique line. It was directed downwards and inwards, lying among the fleshy fibres of the soleus. Although it had a grooved base, no vessel or nerve passed under it. Muscular fibres of the soleus arose from it, and the extremity was tipped with cartilage.

A fuller description of it will be found in the 'Path. Trans.,' vol. xxxiv. No one present at the meeting at which it was exhibited could satisfactorily explain its origin. Fig. 1 shows the position of this process.

MUSCLES.

In the head and neck we found one case of symmetrical slips of muscle with no tendon at either end arising from the front of the first ring of the trachea, and being inserted into the anterior surface of the cricoid cartilage.

FIG. 2.

They were about a fourth of an inch wide and placed immediately on either side of the middle line. Quain makes no mention of these little muscles, the position and extent of which is shown in fig. 2.

The following abnormality is recorded in 'Quain,' 8th edit., vol. i, page 282, and we have met with it once during the present year. Two symmetrical triangular muscular slips passed from the anterior margin of the tendon of the digastric to be inserted into the median raphe, each blending with the fellow of the opposite side and with the mylo-hyoid. They were sup-

plied by the mylo-hyoid branches of the inferior dental on either side. An arrangement something like this is recorded in the 'Guy's Hospital Reports' for 1869, but there the two digastrics blended by their anterior margins, instead of giving off distinct slips as in this case.

Once we found a small muscle about half an inch wide which arose from the upper border of the twelfth rib on the right side, and at its outer part from the upper border of the eleventh. These two heads united to form a thin band, which passed upwards, and was lost on the fascia covering the external intercostal just below the tenth rib. This muscle was about three inches external to the serratus posticus inferior.

On the front of the chest an example of the following muscle occurred. A small tendon arose from the front of the sternum on the right side at the level of the first rib. It passed upwards and outwards and was inserted with the omohyoid into the upper border of the scapula. In its course across the neck it followed pretty closely the course of the supra-scapular artery; so in great part of its extent it lay behind the clavicle.

In the upper extremity the abnormality most frequently observed was a third head to the biceps. Sometimes it was found covering the brachial artery, while at other times that vessel was not overlapped by it.

Twice at least we found the pectoralis minor passing over the coracoid process, and going on to be inserted into the humerus on the outer side of the tendon of the supra-spinatus. The muscles about the shoulder were all well formed, and there was no bursa between the coracoid process and the pectoralis minor. Once this abnormality was present on both sides of the same subject. Quain¹ mentions that the tendon of insertion is not unfrequently detached from the coracoid process, and is carried on by the coraco-acromial ligament to the acromion process or some other neighbouring part. Turner ('Journ. Anat. and Phys.,' vol. xiii, p. 383), "Notes on the Dissection of a Negro," mentions that in the negro which formed the subject of the paper, this muscle had on both sides exactly the arrangement here recorded.

In connection with the pectoralis major, in one body there

¹ 8th ed., vol. i, page 196.

were on both sides muscular slips running parallel to the muscle or loosely connected to it. They arose from the anterior extremity of the sixth rib, and passing upwards were lost on the outer surface of the tendinous origin of the short head of the biceps, sending off processes to strengthen the fascia of the arm from their upper and lower margins. This will be found recorded in Quain, and is not by any means so common as slips from the pectoralis major to the latissimus dorsi. Reeves, although he refers in a general way to extra slips going from the lower true ribs to the arm, does not refer to this variety.¹ Many slips like the one here mentioned have been recorded in the 'Guy's Hospital Reports' for 1871 and 1869, and all writers on abnormalities frequently refer to them.

On both sides of the same subject the following abnormalities occurred. A short muscle arose from the side of the coracoid process, and its fleshy fibres formed a thick bundle which passed over the capsule of the shoulder-joint, being inserted into the humerus, and occupying the whole of the space between the attachments of the subscapularis and teres major to that bone. There was also a third head to the biceps and an extra head of the pronator radii teres arose from the internal intermuscular septum quite distinct from and about half an inch above the rest of the muscle. It passed down and became blended with the mass of the muscle in the middle of its course. In this same subject on one side only there was a special extensor of the trapezium arising from the outer side of the origin of the extensor ossis metacarpi pollicis, and inserted into the back of the trapezium. Also there was a small muscle arising from the fascia covering the back of the carpus, and inserted together with the third dorsal interosseous.

The first of the above abnormalities is very interesting in relation to Wood's views concerning the coraco-brachialis, which he regards as typically composed of three parts, one inserted above the usual insertion, one at it, and one below; and it would seem possible that the abnormal muscle here described was an example of the first part, but very distinct and inserted high up. This abnormality is also referred to in the 'Guy's

¹ 'Human Morphology,' vol. i, p. 100.

Hospital Reports' for 1869, p. 440, where a coraco-brachialis brevis or rotator humeri arising separately from the coracoid process and inserted into the upper part of the humerus is described. The extra head to the pronator radii teres from the inter-muscular septum is not very rare, and if present in cases in which there is a supracondyloid process it may arise from that.¹

The following uncommon abnormality was once observed. Arising from the external supracondyloid ridge and inter-muscular septum by an origin common to it and the extensor carpi radialis longior was a fusiform muscular slip four inches long, from which arose a long narrow tendon running downwards outside the tendon of the supinator longus, and lying on the tendons of the extensor carpi radialis longior, extensor ossis metacarpi pollicis, and extensor primi internodii pollicis; then becoming again muscular, it formed a fleshy rounded bundle which lay parallel to the outer border of the abductor pollicis, and was attached to the base of the first phalanx of the thumb. Whilst it crossed the extensor ossis metacarpi pollicis and extensor primi internodii pollicis, it lay in the same synovial sheath in the annular ligament. It was crossed by the radial nerve in the forearm. This is apparently a variety of Wood's extensor carpi radialis accessorius, but is peculiar in having two fleshy bellies, in being inserted into the phalanx instead of the metacarpal bone, and in going through the annular ligament with the ossis and primi, instead of with the radial extensors.

On one occasion the following unusual position of the œsophageal orifice of the diaphragm was noticed (*vide* Fig. 8). The œsophagus (G) lay to the right of the aorta (A) and almost on the same level; the latter was consequently displaced more to the left than usual, the right crus divided into two pieces, of which the inner formed a band separating the œsophagus from the aorta; there was no decussation between the two crura.

Very few muscular abnormalities were observed in the lower extremity. The one of most interest occurred in a subject in which a portion of the flexor longus pollicis separated from the body of the muscle at the junction of the middle with the lower third, and passed superficial to the remainder of the

¹ Reeves, 'Human Morphology,' vol. i, p. 221.

FIG. 3.

muscle, going through a separate synovial compartment in the annular ligament superficial to that of the long flexor; it then joined the tendon of the flexor longus digitorum, and received the insertion of the whole of the accessorius.

VESSELS.

Connected with the arterial system we have first of all to mention that two or three instances were observed of slightly patent ductus arteriosus, and one or two in which the foramen ovale was not entirely closed. Once the four large vessels (the two subclavians and the two carotids) came off separately from the left side of the arch of the aorta. The two common carotids arose close together in front of the two subclavians, which were separated at their origin by an interval of an eighth of an inch; the right carotid then crossed the trachea obliquely from the sixth to the tenth ring, appearing in the middle line at the upper border of the first piece of the sternum, from which point it gradually acquired its normal position. This very unusual arrangement is not mentioned in Quain; it is, however, allied to an arrangement which occurs naturally among some cetacea, in which the subclavians are

separate vessels, and the two carotids spring from a short stem in the interval between them.

We also noticed the very common variety in which the left carotid comes off with the innominate, and also the condition not unfrequently met with in which the right subclavian comes off fourth in the series of vessels, and passes back to the right behind the œsophagus and trachea, and reaches its natural position after being placed in front of the right carotid. These two abnormalities are so frequently recorded that no references are needed.

Another perhaps not unusual abnormality was that of the internal carotid giving off the occipital three quarters of an inch above the bifurcation; notwithstanding its unusual origin it followed the normal course and distribution of the artery, except that the sterno-mastoid branch was given off separately from the external carotid. In another case the superior thyroid was transferred to the upper part of the common carotid, the crico-thyroid branch arose from the usual position of the superior thyroid, and was the only representative of that artery; this like the previous one is a very common arrangement. The internal carotid was noticed in one subject to make a remarkably sharp sigmoid curve at the level of the atlas, the result of this was that the vessel was an inch and a quarter longer than that of the opposite side.

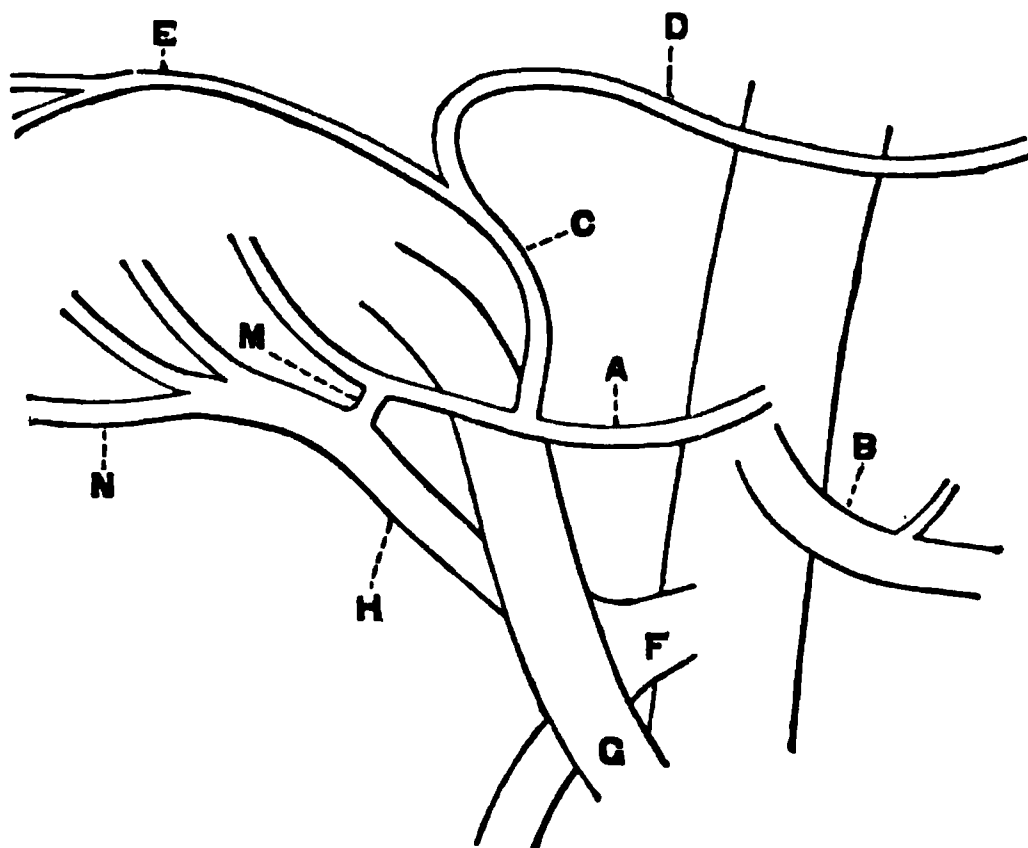
Among several examples of the commoner varieties in the arrangement of the branches of the subclavian, we observed one instance in which the transverse cervical, after taking its usual course over the scalenus anticus, divided in the posterior triangle into superficial cervical, posterior scapular, and two suprascapulars, one going through the notch and the other over the upper border of the scapula half way between the notch and the posterior superior angle. Quain (*op. cit.*) mentions the suprascapular as a branch of the transverse cervical, but makes no reference to the occurrence of two suprascapular arteries.

As in previous years, many instances of the branches of the axillary being given off in one or more groups were noted, and sometimes this took place in subjects in which another not uncommon abnormality, namely, the high bifurcation of the brachial, was present. The highest bifurcation took place

an inch above the lower border of the latissimus dorsi, where the brachial divided into radial and ulnar, in another case it took place two and a half inches above the elbow. The *comes nervi mediani* once came off from the ulnar, and ran as a large branch to the palm of the hand; this is mentioned by Quain (*op. cit.*), and also a better example will be found in the last volume of these reports. It is also mentioned by Reeves (*op. cit.*, p. 282).

We had as usual a large number of abnormal arrangements of the coeliac axis; perhaps the most remarkable of these was the following: the comprehension of this will be aided by reference to Fig. 4. The first branch given off from the

FIG. 4.



abdominal aorta was an artery (A) which arose just above the splenic (B); it crossed over the portal vein giving off a large branch (C) which divided into two phrenic arteries (D and E), of which the left (D) took a long course over the aorta to reach the left side; the artery (A) continued on to supply the liver entering into the portal fissure; the splenic (B) gave off a coronary branch. The next branch (F) came off above the pancreas, and, whilst it lay under the portal vein (G), divided into a large trunk, which took on the supply of both mesenterics, and into a hepatic (H), which had a small communicating artery (M) that connected it with the hepatic, previously described as coming from the aorta; it (H) then divided into

a right and left hepatic for the liver, the cystic (n) coming from the right.

We found one or two examples of a hepatic artery, for the left lobe of the liver, springing from the gastric. This is not an uncommon abnormality, it is mentioned by Quain and is referred to in the 'St. Bartholomew's Hospital Reports' for 1881.

The superior epigastric artery once came forward in the space between the sixth and seventh cartilages, lay on the seventh close under the skin, and then disappeared in the upper part of the rectus. This abnormality was present on the right side only. It is interesting, as when lying on the front of the seventh costal cartilage, the artery is peculiarly liable to injury from being so very superficial.

In an instance in which the internal plantar artery ended in digital branches, it lay under the abductor pollicis and flexor brevis pollicis.

The only venous abnormality that was noticed was the following very interesting one; both lateral sinuses were very small, especially the right, which became quite minute after the exit of a large vein through the mastoid foramen, from this point it was continued on, with a diameter of a sixteenth of an inch, to the jugular foramen. At the outer extremity of the superior petrosal sinus on the right side, three large emissary veins were present; there were also three others opening into the torcular Herophili, one into the commencement of the right lateral, and one into the posterior extremity of the superior longitudinal sinus. All the other sinuses were small. This is a very unusual abnormality; unfortunately we did not take this opportunity of testing the accuracy of the view put forward by Bastian,¹ that the occipital groove on the brain is due to the pressure exerted by the posterior extremity of the right lateral sinus and the right side of the torcular Herophili.

NERVES.

Although there were several slight deviations among the various brachial plexuses examined, there were none that departed sufficiently from the two arrangements usually

¹ 'The Brain as an Organ of the Mind,' p. 401.

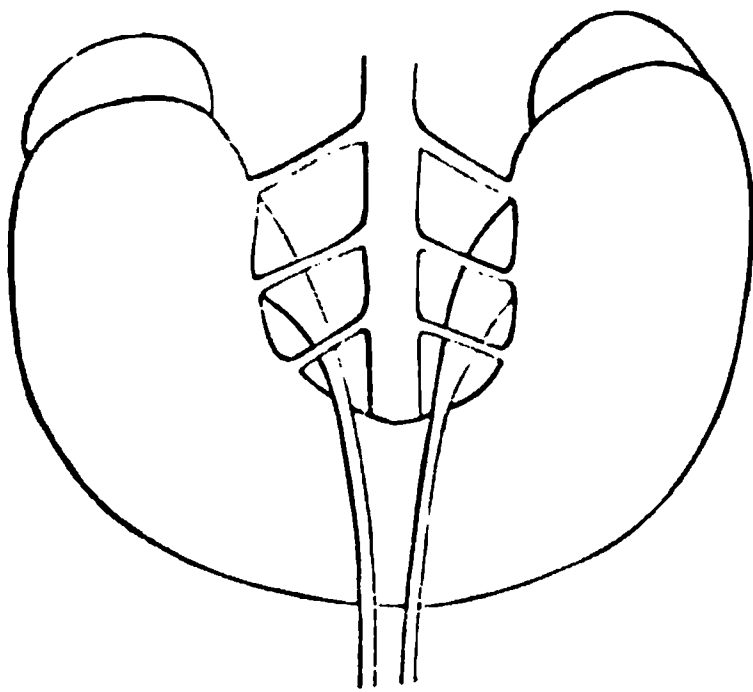
described in the books, to make it worth while to record them.

VISCERA.

As was the case last year, we had one example of a rectum on the right side. The sigmoid flexure was normal, but then the intestine passed across the fifth lumbar vertebra and the first piece of the sacrum to half an inch to the right of the middle line. It then passed vertically down into the pelvis, gradually assuming a median position. The transverse piece of large intestine had a distinct mesentery attaching it to the fifth lumbar vertebra and the first piece of the sacrum.

An interesting example of horse-shoe kidney was noted. The ureters were normal in position, passing down vertically over the arch of communication. There were three symmetrically placed renal arteries, the upper pair being larger than the other two pairs (*vide* Fig. 5).

FIG. 5.



Once we observed several supernumerary suprarenal capsules. This is not very uncommon, and is mentioned by Quain (9th edition, vol. ii, p. 647).

As is customary in the course of a winter session one or two cases of double ureter were observed. In one of them the two ureters did not communicate till within a third of an inch of the bladder.

THE THEORY OF A HEAT CENTRE FROM A CLINICAL POINT OF VIEW.

By W. HALE WHITE, M.D.

THE case of softening of the corpora striata published by me in the last volume of the 'Guy's Hospital Reports,' presented such a remarkable temperature chart, that it seemed worth while to collect a series of cases of nervous diseases accompanied by variations of temperature. This has been done in the present paper, and an attempt has been made to show that they can all be explained physiologically, and furthermore, that they bear out the results which physiologists have arrived at by experiments on animals, for they point to the existence of a cerebral heat-controlling centre.

On looking over the appended cases which have been got together and tabulated, the first thing that strikes one is how few they are. But the reasons for this are many and not far to seek. Firstly, the case is generally so interesting from some other point of view that but little note of the temperature is made at the time of the illness. Secondly, in a paper such as this, if any cause other than nervous is present to account for the rise of temperature the case must be excluded, and for this reason many cases with suppurating bedsores are left out although they had pyrexia which was almost certainly nervous. Thirdly, it must be remembered that if a heat centre exist it would be but rarely that a tumour or hæmorrhage would affect it, for the whole area of the brain is very large and the tumour may be anywhere. Fourthly, the tumour often sets

up meningitis. Having pointed out the reasons for the scarcity of cases I will now consider those I have collected.

As far as I can make out, nervous non-inflammatory pyrexia is present in the following maladies:

1. Tumour of the cord, especially in the cervical region.
2. Tumour of the brain, especially of the pons.
3. Hæmorrhage of the brain, especially of the pons.
4. Embolism of certain cerebral arteries.
5. Ill-defined degenerative changes in the brain.
6. Insular sclerosis.
7. Locomotor ataxy.
8. Obscure nervous cases that die without any change being found post-mortem.
9. Obscure nervous cases that get well. "Hysterical pyrexia."
10. Mental disease.
11. Injury to the spine.
12. Injury to the brain.

Lowering of the temperature is present in the following:—

1. Tumours of the brain.
2. Cerebral hæmorrhage.
3. Degenerative changes in the brain.
4. Mental disease.
5. Injury to the spine.

We will now consider these *seriatim*.

1. *Tumours of the Cord*.—Cases 3 and 4¹ are very good examples of this. It is so rare to get examples of tumour of the cord in which bedsores do not form, that these are the only two instances which I can find in the Reports of Guy's Hospital with good record of temperature. Both these, it is to be observed, are in the cervical region, and pyrexia is a recognised symptom of tumour of the cervical cord; whether or not it is present in a less degree in tumours lower down my tables do not show, and systematic treatises give no information on this point.

2. *Tumours of the Brain*.—Of this cause for hyperpyrexia cases 1 and 2 are very good examples. Cases 5, 6 and 7 did not occur in Guy's Hospital, but as they appeared whilst this

¹ For the cases see the end of the paper.

paper was in progress and are such typical examples, I hope Dr. Bristowe,¹ Dr. Ridge Jones,² and Dr. Beevor³ will pardon my having quoted them at some length. I will take the last one, by Beevor, as an example; a tumour measuring three inches by two and a quarter grew from the upper surface of the cerebellum, extending each side of the middle line; it grew forward between the optic thalami, flattened the corpora quadrigemina, and pressed upon the right optic thalamus, the right crus, and the right cerebellar peduncle; there were no signs of pressure on the pons, medulla or cranial nerves; the temperature went up to 107·4°. Obermier⁴ mentions rise of temperature as one of the terminal symptoms of tumor cerebri but makes no reference to the position of the latter when it occurs.

It will be seen that taking all the cases together we have here six cases of cerebral tumour with hyperpyrexia: in two of these the pons is the only part implicated: in two the tumour was situated between the corpora quadrigemina and the cerebellum, various surrounding structures being more or less implicated: in one there were multiple gummatous nodules around the cerebral vessels and a marked patch of softening in the corpora striata: and in one a gliomatous infiltration of the left hemisphere with affection of the anterior part of the claustrum. This last case is however of less value than the other five, as although the temperature was 103° eleven days before death, ultimately it fell too low to be registered. Putting aside therefore the last case, the other five show that if a case of cerebral tumour have hyperpyrexia the probability is very strong that it is either in the pons or its neighbourhood. Seeing that the tumours in the fifth case were multiple it can hardly be called an exception because there is just the possibility that some small gummatous deposit appreciable only to the microscope was accidentally overlooked.

3. *Cerebral Hæmorrhage.*—In the case of cerebral hæmorrhage the difficulty of selecting good examples is very great, partly because the effect of shock mars nature's experiment, and

¹ Bristowe, 'Brain,' Part xxii, July, 1883.

² Ridge Jones, 'British Medical Journal,' vol. ii, 1883, p. 821.

³ Beevor, 'Brain,' vol. iv, p. 250.

⁴ Obermier 'Ziemssen,' Eng. Trans., vol. xii, p. 263.

partly because the hæmorrhage is usually so extensive as to be of no use for localising purposes. A great rise of temperature is of no importance, from the point of view being considered in this paper, if a previous fall due to shock has been very great, for the rise is then largely reactionary. Case 11 has, however, been recorded as showing one of the greatest if not the greatest range of temperature (viz. from 95.6° to 107.4°) on record. The lowest fall I know of is recorded by Bastian,¹ in whose case 94° was reached and the patient died. Bourneville² gives no range so great as that I have mentioned, but it should be stated that in the case here described the temperature was not taken in the rectum. Allowing, however, that in some cases the thermometric variations are the result of shock in cerebral hæmorrhage, it will not I think be difficult to show that this is by no means the whole of the explanation. Thus the quantity of blood lost is often very small, quite out of all proportion to the changes in temperature that accompany it. For example, there is often not so much loss as a woman suffers in an ordinary confinement or a patient in amputation of the leg. Then there are many cases in which there is no primary fall, and if there be no previous decline of temperature we can hardly explain the subsequent rise by saying that it is the reactionary period after shock. Cases 10 and 12 in the appended tables are examples in point; thus in one the temperature was 100.2° on admission and it steadily rose till death; in the other it was not so high. Then again Bourneville gives many cases in which there was a continuous rise dating from immediately after the accident. Has the locality of the hæmorrhage any constant association with the changes in temperature? This problem presents itself here in a more difficult light than was the case with cerebral tumour, because, as has just been pointed out, the hæmorrhage is either too extensive or else the shock is too great to make the case of any value; but there is one undoubted fact, namely, that temperature rises after effusion of blood into the pons. In proof of this, I can give the following quota-

¹ Bastian, 'Clin. Soc. Trans.,' 1884.

² Bourneville, 'Études cliniques et thermométriques sur les maladies du système nerveux.'

tions; Erb¹ says, "In a case where hæmorrhage occurred in the pons, I observed considerable rise of temperature in the body during the agony. Leyden and others record similar instances; and further, it agrees with well-known experimental facts." Gowers² gives a case of hæmorrhage into the pons in which the temperature rose to 109·2°. Bastian³ says that in bleeding into the pons the temperature may reach 110°. Alexander⁴ gives a case in which a small clot occupied the lower right half of the pons and the temperature was 101·4° in the right, and 102·2° in the left axilla. Wood⁵ also gives other but not very well-marked examples. Wilks,⁶ whilst speaking of hæmorrhage into the pons, mentions a case in which there was a considerable rise of temperature. Bircher⁷ records a case of hæmorrhage into the front part of the frontal lobe, the cerebellum and pons, in which the temperature reached 105·8°. Thus without going further into the literature of the subject we may take it as proved that hæmorrhage limited to the pons, provided that there be no question of shock, causes pyrexia; the exceptional cases will be shown further on to really prove the rule.

With regard to hæmorrhages in other parts we are in a much greater difficulty. Case 10 is a typical one. In it there was a recent clot in the left hemisphere about its middle but not affecting any of the great ganglia. Bourneville⁸ gives the following cases:—Rapid ascent of the temperature to 108·6° in an apoplectiform attack, death in twenty-one hours revealing cerebral hæmorrhage which occupied the right centrum ovale and had opened into the ventricle. Next a very interesting case is given in which the temperature first mounted to 103·4°; then it fell to 100·8° and there were other symptoms of a second hæmorrhage, but again the temperature rose to 107·6°. Here we have very well illustrated the effect of both the hæmorrhagic rise of temperature and the fall due to the

¹ Erb, Ziemssen's 'Encyclopædia,' English Trans., vol. xiii, p. 872.

² Gowers, 'Brain,' vol. ii, p. 466.

³ Bastian, 'Paralysis from Brain Disease,' p. 220.

⁴ Alexander, 'Lancet,' 1875, i, p. 722.

⁵ Wood, 'Fever,' p. 158.

⁶ Wilks, 'Lectures on Diseases of the Nervous System.'

⁷ Bircher, 'Correspondenzbl. für Schweiz. Aerzte,' 1881, Nr. 4.

⁸ Bourneville, op. cit.

shock. Post mortem, there was a hæmorrhage in the left optic thalamus breaking into the ventricle and also one in the right corpus striatum. The case immediately following this is like it, for there was distinct evidence that the hæmorrhages did not take place all together, and on the occurrence of each fresh hæmorrhage there was a fall of temperature although the general tendency was to rise; thus at death it stood at 106.8° . At the autopsy the following were the seats of the hæmorrhage, right corpus striatum, one of the right occipital convolutions, the end of the corpus striatum, the optic thalamus and the cerebral peduncle of the left side, the left superior and inferior cerebellar peduncles. Ollivier¹ gives a case of hæmorrhage into the optic thalamus and crus cerebri with a temperature of 102° , and also one in which both thalami and both crura were affected and the temperature reached 105.4° . Wernicke² records a case of hæmorrhage about the basal ganglia with a temperature of 105° .

So as a result of our investigations as regards hæmorrhage we may say that in the pons, in the neighbourhood of the great ganglia, especially the corpus striatum, or in the centre of the hemisphere above them, it frequently gives rise to a high temperature; when it does not the tendency of the shock to depress the temperature must be borne in mind. Two examples of rise of temperature in cases of old hæmorrhage are given in Charcot's 'Senile Diseases,' but they are too briefly recorded to be of much use for the purposes of this paper.

4. *Embolism*.—As examples of embolism producing pyrexia, the following cases may be cited; one by Gowers³ in which after embolism of the basilar artery the temperature rose to 105.2° evidently owing to cutting off blood supply from the pons; and one by Mills⁴ of multiple cerebral embolism and softening with right hemiplegia, the temperature on the right side being 101° , and on the left 100.2° . The former appears at the end of the paper as Case 14. Both these cases fall in with those we have considered in group three, for in one of

¹ Ollivier, 'Gaz. hebdomadaire,' 12, 1875.

² Wernicke, 'Lehrbuch der Gehirnkrankheiten,' Band ii, p. 48.

³ Gowers, 'Brain,' vol. ii, p. 466.

⁴ Mills, 'Brain,' vol. ii, p. 547.

them the lesion affected the pons and in the other the corpus striatum. Some cases recorded in the next group are probably examples of softening due to embolism.

5. *Ill-defined degenerative changes in the brain.*—By far the most marked example of this is Case 7, which I have recorded in the last volume of the ‘Guy’s Hospital Reports,’¹ in which there was a small patch of softening found post-mortem at the front of each corpus striatum just above the anterior commissure; this was accompanied by secondary degeneration. The temperature rose to 107° or even higher.

Mills gives a case of softening of the pons in which the temperature mounted to 108° before death. In the list at the end of this paper it is No. 9. Bourneville gives the following cases:—Large patch of red softening in the left centrum ovale, a recent patch in the right corpus striatum, the temperature rose to 104.8° before death; in another example there was a patch of red softening in the right hemisphere, the temperature rose to 106.2° ; in another there was softening of the extra-ventricular nucleus with a temperature of 108° ; in a fourth the lesion was rather posterior, namely, on the surface of the sphenoidal lobe, but this is interesting because the temperature only rose to 102° and quickly fell back to normal; next we have two cases in which the temperature mounted to about 104° and there was found considerable softening of the anterior lobe; and lastly, one in which the thermometer registered 105° , with softening in the neighbourhood of the fissure of Rolando. It need hardly be pointed out that here as in the hæmorrhage and embolism groups, the lesion is either in the pons, the neighbourhood of the great basal ganglia, or the brain above them.

6. *Insular sclerosis.*—It is stated that in this disease attacks of pyrexia are liable to come on, but in the four or five cases I have seen these pyrexial paroxysms have been absent. One may take this opportunity of pointing out that the rise of temperature in these cases is not, as Bristowe² says, a means of distinguishing them from hæmorrhage into the brain, for we have already seen that hyperpyrexia is a very frequent symptom of hæmorrhage.

¹ ‘Guy’s Hospital Reports,’ vol. xli, 1882, p. 20.

² Bristowe, ‘Theory and Practice of Medicine.’

7. *Locomotor ataxy*.—In this disease as in insular sclerosis patients are liable to temperature crisis. Case 15 is an example of this. There were three crises in the course of eleven days, in the first and last the temperature rose to 103.2° , in the middle one to 102.4° .

8. *Obscure nervous diseases that result in death with no change found post mortem*.—This is a most interesting class of cases and not so very uncommon. I have seen one or two and heard of others; the only symptom is pyrexia, which may last for weeks; there is absolutely no sign of organic disease, and at last the patient dies, killed as it seems in some cases by the hyperpyrexia. One of those recorded at the end of this paper, viz. Case 19. It will be seen that the woman's only essential symptom for weeks was hyperpyrexia; all other symptoms, such as wasting, weakness, &c., being evidently the result of this; after death nothing was found abnormal in any part of the body. Of course in these cases one must be careful not to overlook unusual causes of hyperpyrexia; for example, as I have shown,¹ an acute inflammatory condition of the medulla which results in death may need the microscope to detect it. Such examples as we are now considering are during life very often, on the theory of probabilities, diagnosed as tuberculosis, and as it is well known, apparently simple pyrexia is often found post-mortem to owe its existence to this cause; nevertheless there is undoubtedly a distinct class of cases whose only symptom is pyrexia, whose end is fatal, and whose lesion is, as far as we can see, undiscoverable.

The diseases in the group we are now considering have been here called nervous because my belief is that probably they are so, and in many the probability is rendered almost a certainty by the other symptoms that are present. It is not excessively rare to come across cases which are diagnosed as "cerebral" or "nervous," and which have hyperpyrexia, but in which on post-mortem examination nothing is found. Case 16 is one in point; one would certainly call such a case nervous, the convulsions were epileptiform accompanied by pyrexia, post-mortem nothing was found. Westphal² has

¹ 'Path. Trans.,' 1881.

² Westphal, 'Archiv für Psychiatrie und Nervenkrankheiten,' Band xiv, Heft i, 1883.

quite recently recorded at great length observations extending over many years on two cases which during life could not be distinguished from insular sclerosis and in which pyrexial paroxysms were occasionally present. In neither of those could any lesion be found post-mortem. There are also cases of hyperpyrexia such as that recorded by Dr. Mahomed, in which the lesion found post-mortem was so completely inadequate to account for the high temperature, that it might be recorded here, but has not been, as one has wished only to mention those cases in which there was absolutely no cause found for the hyperpyrexia. Under this eighth group we may provisionally place puerperal eclampsia, in which the temperature just before the fatal termination is often extremely high; thus Bourneville gives cases in which it went as high as 109°. Later writers follow Braxton Hicks¹ in not considering puerperal eclampsia to be a uræmic condition, and one might here in passing point out that one strong reason for believing it to be distinct from uræmia, is that, in the latter, as was first pointed out by Roberts, the temperature falls. Here one may mention that epilepsy has been said to be a cause of rise of temperature, but in all the cases I have been able to find the rise is so slight that it might easily be explained by the violent muscular movements of the patient. On reference to Clouston's² tables it will be seen how slight the rise is; the temperature is therefore an argument against the theory that puerperal eclampsia is an epileptiform condition, the explanation to which Playfair,³ following Traube and Rosenstein, inclines.

9. *Hysterical hyperpyrexia.*—Many cases which are included under this head probably ought to be only put here provisionally. Of course all the cases included in group eight have a pathology, and probably, although it has hitherto escaped our observation, it is to be found in the nervous system. Now, it is very likely that some of the cases called nervous hyperpyrexia likewise have their lesion in the nervous system, but as they recover they are set down as hysterical. On the other hand there are undoubtedly some cases

¹ Braxton Hicks, 'Obst. Trans.,' vol. viii.

² Clouston, 'Journal of Mental Science,' vol. xiv, p. 34.

³ Playfair, 'Science and Practice of Midwifery,' vol. ii, p. 287.

which have no lesion that we could demonstrate to the naked eye or microscope if we could see the brain, and which therefore ought to be called hysterical or more properly functional. This I say, merely because many accepting Sir James Paget's well known saying, "the patient says, 'I cannot!' the friends say 'You will not!' but the doctor says 'She cannot will!'" would argue that as the will has no influence over the temperature of the body it is hardly fair to speak of hysterical pyrexia; but this is after all a matter only of terms, for if we describe a hysterical patient with contracture of her limbs as having for example a mad motor area in her brain, we can in these cases of hyperpyrexia describe the sufferer as having a mad calorific centre, and we are some way on towards understanding the apparent mystery. Among the cases taken from the hospital records the one which is numbered seventeen in the appendix to this paper is a very good one; it is there seen that the temperature with its accompanying symptoms, such as flushing, &c., was directly under the influence of gold metallo-therapy. Case 18 is also a good example of hysterical pyrexia. There are many other instances in the hospital records which might be and probably are genuine cases of hysterical pyrexia, but for various reasons it has not been thought well to mention them here because there is always the possibility that they were due to some slight ailment or that the temperature was fraudulently produced. Thus there occurs in the hospital reports the very striking case of a hysterical girl who used to amuse herself by producing abscesses in various parts of her body; her temperature was often up, but here there is, of course, the possibility, although from reading the report it is extremely improbable, that the rise was due to the abscesses. The most remarkable case yet recorded of hyperpyrexia is that by Dr. Mahomed,¹ in which the extraordinary height of 128° was reached; the fluctuations were extraordinary not only for their rapidity but for their amplitude, and the variations at the same time in different parts of the body were equally remarkable. There was much controversy about the case at the time, the opinion of the older members of the profession who saw the case was that the temperature was fraudulently produced, whilst the clinical clerks

¹ 'Lancet,' 1881, vol. ii, p. 790.

in charge of it thought it genuine as they were unable to detect any deception. The patient accidentally caught scarlet fever and died, and no explanation could be found post mortem for the hyperpyrexia. In many respects this case was such an extraordinary one that it will not serve us in any way in this paper, and whether it is possible to have such high temperature in the body in such an irregular way must be left to future discovery. In hystero-epilepsy a slight rise of temperature has been recorded, but not more than can be explained on the supposition that the excessive movements are the cause of the rise.

10. *Mental disease*.—A slight rise of temperature may often be detected in the insane; the very full tables by Clouston¹ show this. Even if one had the special knowledge required it would not be within the scope of this paper to discuss questions of insanity, but it is worthy of note that the greatest rise and greatest fall of temperature in mental disease occur in just that one form in which lesions are most constantly found post mortem, namely, general paralysis of the insane.

11. *Injuries to the spine*.—These are a very frequent cause of pyrexia and hyperpyrexia. The most extraordinary on record is that by Teale,² in which the temperature was registered at 122°, but may be said to have reached at least 125°. On reading the record of this case the fact that struck me most was the mention of the young lady's hysterical tendencies, and on referring to the leading article in the *Lancet*³ published at the time I find that the writer is disposed to connect her hysterical symptoms with her hyperpyrexia. So many references are given by Mr. Jacobson⁴ to the best cases of rise of temperature after fracture of the spine that it is quite unnecessary to repeat them here. The temperature in Weber's cases rose as high as 110° and 111° and in Churchill's as high as 106°. The most important points to notice are that the temperature much more often rises than falls after injury to the spine, and that if it is altered at all after injury below the first dorsal vertebra it is only for a rise and never for a fall.

¹ Op. cit.

² 'Clin. Soc. Trans.'

³ 'Lancet,' vol. i, 1875, p. 343.

⁴ Jacobson, 'Holmes' System of Surgery,' 3rd edit., vol. i, p. 654.

12. *Injuries to the brain.*—I have only one case of this, viz. Case 13, and it will be seen how high the temperature ran. The lesion was just where later we shall show that physiologists have placed the heat centre. The chloroform in this case seems to have temporarily brought the temperature down a little.

Before proceeding any further I would remind the reader that many of the rises of temperature which one sees in nervous disorders may be accidental, and not, as some writers think, an essential part of the disease. I remember well the case of a man suffering from insular sclerosis, who had one night an attack of hyperpyrexia, similar to those described as occurring in this disease. We were, however, all disappointed to find that it could be connected with a cigar which the patient, as smoking was forbidden in the ward, had solaced himself by eating. But after making every allowance there remain several cases of pyrexia which can have no other cause than the nervous malady with which the patient is affected.

Having now got together these cases in which nervous disease causes a rise of temperature we will first try to find an explanation, next we will endeavour to explain the cases in which the temperature falls, and lastly we will see if our explanation tallies with physiological facts. Many physicians are extremely sceptical about the influence of the nervous system on the heat of the body, but surely the array of cases one has managed to get together in which the temperature was affected in a purely nervous disease will settle this point; and here one might call attention to the facts that in young children rigors are almost replaced by convulsions, and in adults the rigor is accompanied by severe muscular movements, which facts would receive additional interest if it could be shown that the rise of temperature is of nervous origin. As, however, in the vast majority of pyrexias the probability is that the poison acts directly on the tissues, and it is only in the few that are discussed in this paper that the nervous system is the cause of the rise, we must not be led away by any tempting coincidences which are the more alluring, because, as will be shown later on, the heat centre is situated very near Ferrier's motor areas. There can be but little doubt that rigors and convulsions are only the result of the pyrexia.

I think the cases we have considered may be explained by supposing that there is a calorific centre on the surface of the brain in the region of its middle third, somewhere, that is to say, in the neighbourhood of the fissure of Rolando. We must suppose also that the centre on one side presides over the heat-forming organs of the other, but that owing to the blood circulating so freely through the body the difference in temperature between the two sides of the body that one would expect in cases of injury to one side of the brain, is not always very marked. It is probable that each heat centre is compound; thus at one part of the cerebral surface there is a centre for the heat of the leg of the opposite side, at the other part a centre for the heat of the arm, and so forth; very possibly this view if pushed to its utmost would involve the idea of a nerve-cell or a group of a few nerve-cells presiding over each muscle or each group of muscles or other thermic tissues. Whether or not this is a genuine heat centre one need not now stop to discuss, possibly inasmuch as the muscles are the great heat producing organs of the body it may only be a muscular, vaso-motor one; this is a point which will be returned to later on. Let our theory then be that the centres just mentioned on the cerebral surface are perpetually exercising a restraining influence on the temperature of the body, and that from these centres fibres go to the thermogenetic tissues of the body passing near the central ganglia, down the crura cerebri through the pons, decussating somewhere below that and going by means of the medulla and cord to the tissues.

Now let us look at the cases recorded in this paper from the point of view expressed in the above theory.

Considering first the tumours of the brain and cord, it will be seen that those which are almost if not always accompanied by a rise of temperature are just those which are so situated that it would be almost impossible for them to avoid wounding the heat fibres in their course from the cerebral cortex to the thermogenetic tissues, that is to say those tumours situated in the cervical cord, medulla and pons; in fact so frequently is hyperpyrexia a symptom in these cases that it helps somewhat in localising the tumour. One is aware that the temperature is not always up; thus it is said not to be up in

gliomatous enlargement of the pons,¹ still the number of cases of disease of the pons in which there is a rise of temperature are so many that this will hardly weigh against our argument. Possibly in the few cases of gliomatous enlargement which have had their temperature carefully taken, it has chanced that the calorific fibres have escaped.

With regard to tumours higher up, of which Cases 1 and 2 are examples, case 1 bears admirably on our hypothesis, for the gliomatous infiltration was wedge-shaped with the base of the wedge at the cortex and the apex at the ganglia, and if we suppose several heat centres in the cortex this is just the shape of the degeneration that would be required to catch the fibres in their course from the surface to the central ganglia. In Case 2 there were so many nodules on arteries that they may have stopped the blood-supply to some important calorific centre and hence cut off its habitual restraining influence, or else the patch of softening in the corpora striata may have affected some of the fibres. In Case 5 again, probably the degeneration has just caught some calorific fibres, and so if any one of the cases we have referred to be examined it will be evident that in all of them it is possible for the calorific nerves to have been destroyed, if they take the course we have sketched out; and therefore, if our theory be correct, the restraining influence having been taken off, the temperature rises.

It is evident that the reasoning we have just employed will apply with equal force to hæmorrhages and embolism. See how marked the rise of temperature was in the case of hæmorrhage into the pons and also in that of embolism of the basilar artery, the explanation being that the calorific fibres have been destroyed. In speaking of cerebral hæmorrhage in a previous part of this paper we concluded by saying that "in the pons, in the neighbourhood of the great ganglia, especially the corpus striatum, or in the centre of the hemisphere above them, it frequently gives rise to a high temperature." But these are just the positions in which we saw cerebral tumours giving rise to pyrexia, so may we not conclude that in hæmorrhage as in tumour this pyrexia is due to destruction of the calorific inhibitory fibres?

¹ Money, 'Medico-Chirurg. Trans.,' vol. lxvi, p. 291.

In the cases of ill-defined degenerative changes it is interesting to observe how in the very case in which the degeneration was furthest removed from the supposed course of the calorific fibres, namely the one in which it was situated in the sphenoidal lobe, the rise of temperature was slight and transitory. In the case previously recorded in these reports we must suppose that the degeneration extending from the corpora striata to the lumbar cord on both sides, in some part of its course affected, by spreading, the calorific inhibitory nerves. This is borne out by the fact that the two patches were situated in the corpora striata, and we have seen reasons for believing that the calorific fibres descending from the cortex pass near these bodies. I think, as we have previously come to the conclusion that degenerative changes which are accompanied by hyperpyrexia are most frequently situated in the same position as hæmorrhage and tumours having the same symptom, we may conclude that the hyperpyrexia in both cases is similar, that is to say, due to implication of the calorific fibres.

With regard to the rises found in insular sclerosis and locomotor ataxy one has only to suppose that the patch of sclerotic change happens to intercept one of these calorific nerves. Dr. Buzzard¹ has already expressed his belief that the gastric crises may be explained by affection of the gastric centre in the medulla; surely if this be so it is not irrational to attribute the temperature crises to a similar cause. It will be urged, would not the effect of such a patch be to irritate some of these fibres and thus cause a fall of temperature, before it got to the stage of destruction? Probably it does, but the fall is never noticed because it is slight. This is not difficult to understand, for supposing as we must, considering the great height to which temperature often rises when the supposed restraining influence is destroyed, that there is always a very strong inhibitory influence passing to the tissues to restrain their metabolism, it is evident that the irritation of a few of these fibres will have but little further inhibitory influence, whilst their destruction would cause a considerable rise of temperature. It is more difficult to understand why these rises ever pass off, but it is hardly more extraordinary than many similar problems. For example, why do the lightning pains come on in paroxysms?

¹ 'Clinical Lectures.'

It is to be noted that if the crises are extremely severe they do not pass off; thus a termination accompanied by an attack of hyperpyrexia is one of the endings of insular sclerosis mentioned in the text-books; possibly too, in the slighter attacks the destruction is so slight that some other part of the brain can take on the work of the destroyed part. I am inclined therefore to think that the occasional presence of crises in locomotor ataxy and insular sclerosis supports the theory of calorific centres in the brain with efferent nerves proceeding from them.

The eighth group of nervous hyperpyrexias in which nothing is found post-mortem can, it seems to me, be only satisfactorily explained by the theory of a heat centre, to which indeed it lends much support. Thus to explain these cases we should have to say that there was a sclerotic change affecting the heat centres or the fibres proceeding from them, and that this change was visible only to microscopical examination. Surely this is not an extravagant explanation; it is common enough to find changes which are only recognisable on microscopic examination. It will be urged that it is very unusual for a sclerotic change to affect a single centre in that way; no doubt it is, but we know of such cases, as for example, glosso-labio-laryngeal palsy. Moreover, these cases of hyperpyrexia are themselves excessively uncommon. It is not necessary for the calorific centres themselves to be affected, destruction of the nerve-fibres proceeding from them will have a like result. These cases of obscure nervous pyrexia die from the exhaustion produced by the long-continued fever.

Two explanations are possible in the ninth group, that of hysterical pyrexia. One is that there was an actual lesion and that the patient recovered owing to the function of the part of the nervous system destroyed being taken on by some other part; and another, which is more likely, is that the sufferer had a mad calorific centre, just as in hysterical hemiplegia she may be supposed to have a mad or perverted motor area. Sure it is that the temperature is not under the control of the will, but that is no reason why its nervous mechanism should not be liable to hysterical vagaries. The well-known saying "She cannot will!" before referred to, does not by any means cover the whole ground, if by it is meant that only

those powers over which the will has control can be perverted by hysteria, for no person can will to produce a phantom tumour, but nevertheless it is a hysterical symptom. If the phrase means that the patient has not the will to remove the hysterical symptom, then likewise it loses its force, for most hysterical symptoms that pass off are not removed by the will, but by something happening to the patient which occupies her whole mind and, so to speak, drives out the hysteria; but were the disease inability to will we should expect her to recover by willing to remove her symptoms. In short it is a very happy saying which is applicable only to a part of the disease hysteria and therefore cannot be taken to invalidate our supposition that the temperatures we are at present considering are really hysterical. Nor must the fact that girls suffering from hysterical pyrexia sometimes get bed-sores from pressure, and waste considerably from the increased temperature, be allowed to disprove it, for it is well known that hysterical girls have allowed the leg to be amputated for a hysterical pain. Although no doubt difficult to understand fully, these cases of hysterical pyrexia seem to me to lend considerable support to the theory of a heat centre, for they are totally inexplicable in any other way; but if we imagine that these patients have a mad calorific centre we have a fairly simple explanation.

As in the ninth so in the tenth group two explanations are possible, one that an actual lesion has affected calorific centres or fibres, and another that the action of the calorific centre is perverted; the second alternative is not so likely to be of service here as it was in the last class of cases, for it is just in the cases in which a demonstrable pathology is present, viz. in those of general paralysis of the insane, that pyrexia is most often present also. On the theory which is being urged in this paper this would be due to the implication in the sclerotic change of some calorific centre or nerve tract. It is well known that the attacks of pyrexia, to which general paralytics are liable, are often transient, as they are in the cases of insular sclerosis and locomotor ataxy, but whilst mentioning these diseases we considered these difficulties and therefore need not revert to them here.

With those cases in which after injury to the spine the

temperature rises, it is so obvious that the same explanation which served in the cases of tumour of the cervical cord will aid us here also, that we need not refer to it.

We have now shown how all the cases of nervous hyperpyrexia we have recorded or referred to can be explained by the existence of cortical calorific centres, with fibres down which inhibitory impulses are always passing proceeding from them to the thermogenetic tissues. Tetanus and hydrophobia have not been considered because of the strong probability that they are not genuine nervous diseases but owe their origin to some poison.

The next points to be considered are, firstly, why in some nervous cases does the temperature fall? and secondly, does this invalidate the theory we have put forward for the rise of temperature?

1. *Tumours of the Brain.*—The examples of tumour of the brain causing a fall of temperature are few, much fewer than those which cause a rise. In Case 1 the temperature after being 103° fell before death till it was too low to be registered; it will be noted in this case that the cord was affected also.

2. *Cerebral Hæmorrhage.*—Here we are in no difficulty to account for a fall which is often very great; all who have written on the subject are agreed that it is due to shock. We have already mentioned a case recorded at the end of this paper, in which the fall was excessive and was accompanied by a very marked reactionary rise. When there is no fall but in its place a rise, we must suppose that the shock has not been sufficient to counteract the rise due to the injury to the calorific nerves or centres. Bastian believed, when he recorded his case of extremely low temperature after cerebral hæmorrhage, that it was the lowest on record, but I have found one still lower recorded by Erb¹ in which the temperature fell so extraordinarily low as 87.8° .

3. *Degenerative changes in the Brain.*—This is a vague class; many of its cases are examples of a sclerotic condition, but for convenience of arrangement they have all been put together in one group. Erb in the article just cited gives an example of "sclerosis of the brain affecting especially the left side," in

¹ Erb, 'Deutsch. Arch. für klin. Med.,' Band i, p. 180, "Ueber die Agoniesteigerung der Körperwärme bei Krankheiten des Centralnervensystems."

which the temperature fell to 89.7° . Another case in which it sank even as low as 84° is recorded by Greenhow,¹ and Otto Hebold also gives cases, but they are not described with much accuracy and the account of the post-mortem is insufficient. All one can say with reference to this class of cases is that there are examples occurring occasionally in which a chronic inflammatory, or sometimes perhaps purely degenerative change affects the brain, which is accompanied by a fall of temperature. The point to which one would here direct attention is the large extent of surface implicated.

4. *Mental Disease.*—As in the case of rise of temperature so here, one must be a specialist in mental diseases to have sufficient knowledge to discuss these cases; the chief point worthy of note is that it is in general paralysis of the insane that the greatest fall is present.

5. *Injury to the Spine.*—Cases of low temperature following injury to the spinal column will be found recorded in Mr. Jacobson's article already mentioned, where references to other cases will also be seen. It is well to bear in mind Mr. Jacobson's footnote, "As far as I am aware no case has been published of excessive lowering of temperature after any injury below the first dorsal vertebra."

Before proceeding to discuss these cases of lowering of temperature, one would first of all point out that, we must bear in mind the existence of a vaso-motor centre in the medulla the exact position of which has been made out by Owsjannikow. Now from this centre fibres run down the cord to all the arterioles in the body, so that supposing the hypothesis we have already put forward of calorific nerves coming from the cerebral cortex to be correct, it is evident that we have two sets of nerves below the medulla, viz. the vaso-motor and the calorific. When we come to consider the physiological aspect of the question we shall see that widespread vaso-motor palsy means fall of temperature.

In all the cases we have mentioned there are four possibilities any one of which taken separately or any two, or three, or all combined might cause the fall. Thus the vaso-motor system might have been influenced so as to cause a wide-spread vaso-motor palsy; secondly, shock may have caused the

¹ 'Clin. Soc. Trans.,' vol. iii, p. 164.

lowering of temperature ; thirdly, the whole lowering of vital functions may have been so great owing to the extent of the lesion that the temperature fell in consequence ; or lastly, the new growth, hæmorrhage or injury may have only been sufficient to irritate and not destroy the calorific inhibitory fibres and thus the temperature has fallen.

Applying this to the first group, that of tumour of the brain, seeing in the case recorded how extensive was the new growth affecting as it did both cord and brain, it seems to me very probable that it may just before death have begun to influence some vaso-motor fibres in the cervical cord, but that this affection, not having gone far, was undiscoverable post-mortem ; this is borne out by the fact that the temperature did not begin to fall till just before death. Or again the whole vital condition of the patient may have been so lowered that the temperature fell in consequence. The fourth of the above suggestions is hardly probable in this case because the destruction of the healthy cerebral substance was too great. We have so often referred to shock being the cause of the fall of temperature in cerebral hæmorrhage that we need not do so again here.

In that obscure class of cases we have grouped together under the single name degenerative, it is evident that any one of the four causes we have put forward above may have been at work. Thus the vaso-motor centre may have become implicated, and here one would suggest that it would seem better for many pathological reasons to suppose that this centre is double, or even more compound, for seeing the great importance of the skin in the regulation of the temperature of the body, it would be a great convenience to the economy if it had a separate vaso-motor centre to itself ; if so, possibly in some of these cases of extremely low temperature this centre alone is affected. The way in which the remaining three supposed causes for a fall of temperature might be made to apply here is so apparent that no time need be wasted in pointing it out. In those cases of the mental disease group in which there is an obvious lesion, such as general paralysis, it is quite within the realms of probability that the change has affected either the vaso-motor centre, or that it has irritated the inhibitory calorific fibres.

Next let us take the group of cases of injury to the spine. Fall of temperature after fracture of the cervical spine is the exception, the rule is a rise. Hutchinson¹ records a case in which it fell in the urethra to 93° and in the rectum to 95.8° , and no rise took place before death; Nieden² gives a case in which the temperature fell to 80.6° . In the first of these two cases the fracture was at the fifth cervical vertebra, in the second at the first dorsal. Now we have already seen that injury to the cord in the cervical region usually causes a rise of temperature, in fact there is a case recorded by Churchill³ of fracture of the fifth cervical vertebra in which the temperature rose to 110° . Thus we have two cases of fracture at the same level, in one the temperature falls to 95.8° , in another it rises to 110° , a range of 14.2° . Other examples of fall might be mentioned, but it is such a well-known fact that we need not do more than take the above as typical examples. Hutchinson does not attempt to explain either the rise or the fall by calorific nerves, but accounts for the fall by supposing that the blood circulating through the paralysed parts gets cooler owing to the cessation of "the functional activity" of the paralysed parts. But surely one of the functions of the muscles is to produce heat, they are the great thermogenetic tissues of the body; this is a distinct function apart from that of motion; if not imagine the disastrous results that would follow when one lies quietly asleep, for in spite of the most delicate regulative mechanism the heat produced by the movements of respiration and of the heart could hardly be so husbanded as to keep the temperature normal during a long sleep. If instead of "functional activity" Hutchinson had said thermogenetic functional activity, he would probably have been correct, but as it reads there is no evidence to show that he looks upon the muscles as having any other function than that of motion, and thus one is led to conclude that he regards the fall of temperature as due to the motor paralysis. He then goes on to explain the numerous cases in which it rises by supposing that in these the heart's action is more powerful than in those in which it falls; but surely this cannot

¹ 'Lancet,' vol. i, 1875, p. 713, *et seq.*

² 'Clin. Soc. Trans.,' vol. vi, p. 75.

³ 'St. Thomas's Hosp. Reports,' vol. i.

be a sufficient explanation. The lowest temperature recorded after fractured spine is about 80° and the highest is over 110° , so that the range is at least 30° , and all this is said to be due to increased vigour of the heart. If this were so, variations of say 5° at least due to this cause would be features of every day occurrence, for the force and frequency of the heart's action are very inconstant: but such variations are against all experience. It seems to me that neither of Hutchinson's suggestions, namely the referring the fall of temperature to the paralysis and the rise to the vigour of the heart's action, are tenable, for as just stated, if they were, every time one was very quiet as from deep sleep the temperature would fall, and a slight accidental increase of the vigour of the heart would send it up. Then, again, in referring the rise of temperature to the increased cardiac beat and its fall to diminished cardiac beat is there not a confusion of cause and effect? It seems to me the reason why in the first case the heart's beat goes up is that the temperature rises, and the reason why in the second cases the beat is less strong is that the temperature falls. One is, of course, aware that Marey has stated that diminished arterial pressure leads to increased cardiac beat; if this were so, in consequence of the widespread vaso-motor palsy resulting from fractured spine, the cardiac beat would be increased because of the diminished arterial pressure. This question need not be discussed, as there is every probability that Marey is wrong.¹ As previously mentioned, the rises of temperature are probably due to the injury to the calorific inhibitory nerves going from the cerebral cortex to the thermogenetic tissues, and various reasons may be found for the fall in some cases of fractured spine. Thus if the accident by chance injure the vaso-motor nerves going from the centre in the medulla considerably more than it does the calorific inhibitory nerves then we shall get a fall, the extent of the fall or of the rise depending upon which fibres are most injured; it may so happen that both are injured in such proportion that there is neither a rise nor a fall. In some cases, but probably not many, shock may cause the fall, and in some the injury may be of such a nature that the fall is due to irritation and not to destruction of the calorific inhibitory nerves. We now

¹ Burdon Sanderson, 'The Process of Fever,' part ii, p. 9.

see how it is quite possible for an injury at the same level in one case to raise and in another to depress the temperature, the result depending entirely upon the strands of fibres which are most affected by the injury; it is not more remarkable than that carcinoma about the portal fissure should in some cases produce jaundice, in others ascites, and in others both, depending of course on the degree to which the biliary ducts and portal vein are implicated.

The twelfth group of injury to the brain is most important, for here we have a lesion of the brain without any inflammation producing hyperpyrexia, although only the grey matter was affected; there seems to me no way of explaining this case, except on the theory of a heat centre. The centre having been destroyed the temperature rose; it is an exact counterpart of physiological experiments, to be described later on.

This completes the second part of the paper; we have now tried to show that the cases of nervous hyperpyrexia may be explained on the assumption that there are calorific inhibitory nerves going from the cerebral cortex to the muscles and glands, and that the fact that sometimes the temperature falls does not invalidate this theory. Next we must see if this accords with the physiological side of the question.

Wood's¹ paper is the most able and recent exposition of the view which holds that there are calorific nerves. His researches extended over many years and he made over two hundred experiments. The chief objection to them is that the liabilities of error with the calorimeter and in the calculation of the specific heat are rather difficult to avoid; in fact, in man, as Burdon Sanderson in the paper above quoted has pointed out, the amount of perspiration is such as to make calorimetrical observations² of but little value; still, as Wood used dogs, and he made a large number of experiments, his results may be taken as tolerably accurate. His results may be thus formulated:

¹ H. C. Wood, 'Fever: Smithsonian Contributions to Knowledge,' 857. Philadelphia, 1880.

² Those interested in physiological calorimetry will find a paper by Bevan Lewis in the 'West Riding Asylum Reports' for 1876 on "Calorimetrical Observations on the Influence of various Alkaloids on the Generation of Animal Heat."

1. Section of the spinal cord high enough to produce a wide-spread vaso-motor paralysis causes first a fall, then a rise of temperature. The extent of the fall depends largely upon the temperature of the surrounding atmosphere; thus, if the animal be wrapped in cotton wool the fall is slight, but if the room be very cold the animal quickly dies. The temperature of the skin does not fall quite so much as that of the interior of the body.

2. Owing to the vaso-motor paralysis, the superficial vessels of the skin are unable to contract, hence heat dissipation is increased proportionally to the difference in temperature between the animal and the external atmosphere.

3. Later on the hourly heat dissipation is diminished.

4. But this diminution of heat dissipation means, inasmuch as the temperature of the animal remains at normal or below it, that there is diminished heat production.

5. If the effect of the cooling of the body be done away with, sometimes there is a diminished production of heat, sometimes, especially in robust animals, an increased production.

6. Both the last statement and also experiments show that the diminished heat production is due to the cooling of the body diminishing the rapidity of chemical change.

7. If correct, these conclusions show that on section of the cord two factors are at work, one a vaso-motor which tends to depress the bodily temperature, and another which often tends to raise it.

One cannot go farther without pointing out how exactly this tallies with the conclusions we came to from clinical facts, for from them we argued that there were two sets of fibres in the cord, one a calorific inhibitory proceeding from the cerebral cortex, and another a vaso-motor set proceeding from the centre in the medulla: on injury to the cord, sometimes, owing to destruction of the calorific, the temperature rose; sometimes, owing to vaso-motor paralysis, the temperature fell. If it be argued that the temperature does not fall quite so often in man as in rabbits we must remember that in man the external temperature is usually kept high by means of hot-water bottles, blankets, &c., and also that it behoves one to be cautious how one argues too freely from one kind of animal to another in minuter details. We have an example

of this in the different results obtained concerning cerebral localisation in dogs and monkeys respectively.

The statement that I have numbered 4 seems to me to be open to question. It is a well-known fact in physics that when a hot body is cooling to the temperature of a cool medium the rapidity with which it loses heat diminishes the nearer it gets to the temperature of the medium; yet Wood makes a strong point of the fact that a certain number of hours after the section the hourly dissipation of heat is less than it was immediately after the section. Surely, as the animal is slowly cooling, this is what one would expect, without its necessarily meaning that the production was less. Direct experiment goes, however, to prove what this argument fails to do. But although it is hardly a matter of vital importance in the question under consideration whether or not there is later on a diminished heat production, it is just what one would expect, unless the animal be kept artificially warm; for not only will the cooled blood returning from the surface check the metabolism of the tissues, but the circulation itself will be more sluggish. We will continue with Wood's steps in reasoning out the subject. Having come to the conclusion that in section of the cord there are two factors at work, one tending to depress the bodily temperature and another to raise it, he made a series of experiments the results of which may be set out in the following form:

1. Sections made below the junction of the pons and medulla caused a great paralysis in the general vaso-motor system, because after section anywhere below that point stimulation of a sensory nerve failed to cause any rise of the blood pressure in an animal which had been quieted by curare and in which the vagi had been severed.

2. Sections at the junction of the pons and medulla caused no alteration in the general vaso-motor system.

3. Therefore the general vaso-motor centre does not extend higher than just below that point. This confirms the experiments of Owsjannikow upon the position of the vaso-motor centre.

4. Section of the medulla anywhere below the junction of the pons and medulla caused a great fall of temperature, and "thus confirmed the belief that the fall was due to vaso-motor

paralysis." By these sections the heat production was decreased and there was primarily increased heat dissipation.

5. Section at the junction of the pons and medulla caused a great rise of temperature. The cases in which this does not take place are probably to be explained by the fact of the bleeding into the fourth ventricle, the clot pressing on the vaso-motor centre, or else by the fact that the parts are so small that it is impossible to sever the medulla from the pons without wounding the vaso-motor centre.

6. Section at the junction of the pons and medulla is followed by an increased heat dissipation and increased heat production. The increased dissipation usually not keeping pace with the increased production, the bodily temperature rises. The cases in which no rise takes place are not really exceptional, but only instances in which heat dissipation is increased proportionally to or faster than heat production, so that no rise of temperature occurs.

7. The increased dissipation is merely a result of increased production, a warmer body naturally giving off more heat than a cool one.

We may I think say that the above physiological statements are in accordance with our clinical facts. Thus we have mentioned several well authenticated cases of fall of temperature after injury to the cervical spine; but with lesions higher up, if we put aside cases in which shock is present and likewise those in which there is, at the same time, an affection of the cord or medulla, the cases of fall of temperature after cerebral disease or injury are extremely few.

Why in some of Wood's experiments the dissipation increased as rapidly as the production, and in others it did not, may seem at first sight strange, but on consideration it is seen that many factors would affect this, for example, the temperature of the surrounding atmosphere, the coat of the animal, &c.

The next question discussed by Wood is, why does section at the junction of the pons and medulla cause these changes? Is it because of the irritation of the contiguous vaso-motor centre as believed by Bruck and Günter? Wood thinks not, for firstly, when needles are plunged in at this point, the temperature rises and he attributes this rise not to irritation of

the contiguous vaso-motor centre but to paralysis owing to their section of nerve-fibres which pass to the body from the brain above ; secondly, the rise comes to its height some time after the plunging in of the needles, but were it irritative its effect would be most felt early in the experiment ; thirdly, a slight wound in this situation caused great diminution of heat production together with fall of temperature ; the wound in this case was so high up that it could not have caused vaso-motor palsy, and therefore he concludes that this diminution was due to irritation of the inhibitory nerve-fibres in the pons. Wood, therefore, believes that " the rise of bodily temperature and heat production following upon separation of the pons from the medulla is paralytic and due to the removal of some active force."

Wood's conclusion is practically the same as we arrived at from a clinical point of view, for we thought the hyperpyrexia was due to irritation of the inhibitory calorific nerves, that is to say, paralytic and due to the removal of some active force. It is to be regretted that in this part of the argument only one satisfactory experiment, viz. that of a slight wound by section of the pons, is given, because if the needles be supposed to divide the fibres it would be open to a critic to say that the small wound had had the same effect ; possibly the fibres run in the centre of the pons and the small wound only went far enough to irritate them while the needles were in just the position to divide them. Considering the smallness of the parts operated upon and the difficulty of the operation it would be wise not to lay too much stress on that single experiment.

Next Wood points out that it follows from what has gone before that a centre for the control of the heat of the body exists higher up than the junction of the pons and the medulla, and suggests that it is either a vaso-motor centre which presides solely over the blood supply of the muscles or else a genuine heat centre. He then adduces the following confirmation of this view.

Heidenhain¹ has shown that irritation of a sensory nerve causes a fall of temperature of the whole body. This, as Wood remarks, cannot be due to a change of blood pressure because

¹ ' Pflüger's Archives,' 1870, p. 504.

it occurs in parts from which the blood supply is cut off, and furthermore the fall persists for some time after the sensory stimulus is applied and long after any effects on the circulation must have ceased. This seems to show that there exists a heat centre which is capable of being inhibited by a sensory nerve stimulation, and this centre, for reasons formerly adduced, is probably above the junction of the pons and medulla. If this view be correct, after separation of the pons from the medulla stimulation of a sensory nerve ought to cause no fall of temperature although it would influence blood pressure. This was found to be uniformly the case, and therefore it is taken as proved that there is some centre situated in or above the pons, controlling the temperature of the body. This centre may be vaso-motor for the muscles, for they perhaps do not contain enough blood to affect blood pressure, or it may be a genuine calorific centre.

Then follows a description of several experiments in which the attempt was made to discover whether or not this centre was situated in the pons, and to determine this, irritating fluids were injected into that body; but such an experiment has only to be mentioned in order to show its uselessness, the lesions produced must be far too coarse and gross to produce any reliable results.

Tscheschichin¹ was, I believe, the first to make experiments showing that the brain had any influence on the heat formation of the body. His results were warmly combated by Lewizky and Bruck and Günter. Copious references to German authorities will be found in an article by Riegel.² Later than this Murri³ has shown that the nervous system has probably a genuine calorific influence. Eulenberg and Landois⁴ found that in dogs destruction of a portion of the brain behind the sulcus cruciatus increased the temperature of the opposite extremities, and that the centre for the arm

¹ Tscheschichin, "Zur Lehre von der thierischen Wärme," Reichert and Du Bois-Reymond's 'Archives,' 1866, and "Zur Fieberlehre Deut. Archiv für klin. Med.,' Band ii, 1867.

² Riegel, "Ueber den Einfluss des centralnerven Systems auf die thierische Wärme," 'Pflüger's Archiv,' Band v, 1872, p. 629.

³ Murri, 'Del potere regolatore della temperatura animale,' Florence, 1873, and 'Sulla teoria della febbre.' Fermo, 1874.

⁴ 'Virchow's Archives,' Bd. lxxviii, p. 245.

was in front of that for the leg; irritation of the same parts caused a fall of temperature. Hitzig¹ has confirmed these results, and Wood has made fourteen experiments in which he shows that "in the dog destruction of the brain region known as the first cerebral convolution posterior to and in the vicinity of the sulcus cruciatus is followed by an increase of heat production." If both sides are injured the increase is greater than if only one is injured. One or two experiments are given which go to show that irritation of the same region causes a diminished heat production.

Next it was found that in an animal in which the vagi and splanchnics were cut stimulation of a sensory nerve, by acting on the small vessels whose sympathetic was not destroyed (*i.e.* chiefly muscular ones), caused a rise in blood pressure; and destruction of the cortical region above indicated did not affect this pressure either way, therefore it is concluded that this centre is not vaso-motor for the muscles.

A single experiment is then given to show that after separation of the pons and medulla and division of the splanchnics and vagi, blood pressure still rises on stimulation of a sensory nerve; from which it is concluded that the muscular vaso-motor centre exists below the junction of the pons and medulla.

The last two experiments having proved conclusively that the calorific centre in the neighbourhood is not muscular vaso-motor, the only option left to us is to conclude that it is a genuine calorific centre presiding over the thermogenetic chemical changes in the thermogenetic tissues.

With regard to the position of this centre, the clinical facts are fully confirmed by physiology. We came, in the former part of this paper, to the conclusion that the calorific centre was situated about the middle of the outer cerebral surface: but that is in the very part where Hitzig, Wood, and others, have localised the heat centre in dogs, so that we may, I think, say that the results of our attempt to localise the heat centre by clinical observation are fully confirmed by the latest physiological experiments. The physiological part of this paper was prepared from Wood's and other books at least six weeks before the clinical part, and then the two were compared with the above result, so that one is quite innocent of any attempt

¹ Hitzig, 'Centralblatt für die med. Wissensch.,' 1876, p. 323.

to stretch points in order to make the two parts agree. The case to which I would particularly direct attention is that of a bullet wound.

Our cases do not help us much in proving whether or not the centre is muscular vaso-motor or genuinely calorific. Rosenthal,¹ who is perhaps the greatest authority on this subject, withholds his opinion. He quite admits that injury to the cerebral cortex in dogs in the region of the sulcus cruciatus does affect the temperature, but how he is not prepared to say. Thus he concludes, in his notice of Wood's experiments in the 'Handbuch,' by saying, "all these observations show with certainty the influence of the brain upon the local and general temperature according as the same is dependent upon a vaso-motor influence. But a direct influence upon heat production may depend upon this, in spite of the positive facts of Wood's, which are not yet received as certain." It would perhaps be presumptuous for any one not much versed in physiological experiments to give an opinion, but still it certainly does seem that Wood's evidence in favour of a true heat centre is very strong, especially when coupled with the fact that stimulation of nerve going to a part will raise its temperature, even if the blood supply be cut off. A quarter of the whole blood of the body is in the skeletal muscles: now, if the muscular vessels were so dilated as to take to those organs enough blood to raise the temperature of the whole body ten or fifteen degrees, surely we should expect some marked effects due to great lowering of arterial pressure in other parts. But of this we have no evidence. In the nervous pyrexial cases now under consideration, they should be examined in future more in detail to determine the blood pressure; but if it were found to be diminished it would by no means prove that the rise of temperature was due to more blood going to the muscles, and it must be remembered that if this were so we should expect the legs for example to swell, to be particularly liable to hæmorrhage and inflammation, &c. True it is that the secretion of urine in fever is much lessened, but not more than, one would suppose,

¹ Rosenthal, 'Zur Kenntniss der Wärmeregulirung bei den Warmblütiger Thiere,' Erlangen, 1872, and Hermann's 'Handbuch der Physiologie,' Band iv, Theil 2.

could be explained by the more rapid evaporation from the skin owing to the increase of temperature; in fact, considering that this alone must much diminish the quantity of the urinary secretion we should, were the muscular vaso-motor theory correct, expect to find absolute suppression of urine in nervous pyrexia. Of late, especially since Charcot's discoveries, so much new light has been shed on the subject of trophic nerve influence, and we have so many examples of nerves having a direct influence over tissues, as seen, for example, in acute bed sore, anterior poliomyelitis, herpes, anæsthetic leprosy, perforating ulcer of the foot, &c., that, reasoning by analogy, it seems only probable that there should be genuine calorific nerves. Lastly, quinine and salicylic acid, both of which powerfully reduce the temperature of the body in fever, are not generally accredited with any power to make minute vessels contract. So that whilst we cannot consider the question finally settled, the balance of evidence seems strongly to favour the opinion that the centre on the cerebral surface is a genuine calorific centre, and not simply a muscular vaso-motor one. Quite recently a case has been published¹ in reference to which Dr. Burdon Sanderson says: "The pyrexia of your patient I take to be dependent upon a disorder of that part of the central nervous system which regulates most chemical processes by which heat is produced." Thus we see that that distinguished physiologist agrees with us that pyrexia may be due to disorders of the central nervous system.

Appended are the cases that have been referred to whilst writing the above paper.

CASE 1.²—Thos. P—, æt. 11, schoolboy, admitted into John Ward under Dr. Pye-Smith, July 11th, 1883.

Family history.—Mother has had two miscarriages.

Personal history.—Had chorea two years ago. Has always been left-handed.

Present illness.—For the last four weeks has suffered from headache and listlessness. Ten days ago became suddenly

¹ Withers Moore, "On the Production of Heat in Fever," 'Brit. Med. Jour.,' Feb. 9, 1884, p. 258.

² For the temperature in this case, see the chart at the end of the paper.

blind in the left eye, the right hand also became weak, and in a few days was powerless. Five days ago the right leg became weak, and in three days was completely paralysed. During the last four days "pins and needles" in the right upper and lower extremities. Some sickness.

On admission.—Temp. 101.4° . Heart and lung signs normal. Pupils react to light. Mental faculties good. Right optic disc normal, left optic disc grey, blurred. Paralysis of right face, upper and lower extremities. "Pins and needles" in both extremities. Paroxysmal pain in right thigh. Reflexes: right plantar normal, cremasteric and abdominal absent, patellar exaggerated, ankle clonus well marked; left all normal except that abdominal is absent and slight ankle clonus is present. Speaks well. Sees well with right eye, left blind.

July 13th.—Inability to micturate, catheter used. Pain in right leg. Right axilla and right popliteal space 3° hotter than left.

14th.—Drowsy and cannot speak well. Passes motions under him.

15th.—Left leg, which has been getting weaker, is almost powerless.

16th.—Tongue deviates to right side, all reflexes abolished except abdominal. Pupils widely dilated, left does not respond to stimulus of accommodation, but slightly to that of light. Pain in left leg, no anæsthesia or hyperæsthesia.

17th.—Electrical report: faradism, all muscles excessively irritable, especially right; galvanism normal. Anaesthesia and anæsthesia in legs and feet, these two symptoms diminish from below upwards.

19th.—Rigidity of right arm. Pupils of normal size. Pain on pressure over anterior inferior angle of left parietal bone.

20th.—Complains of headache and pain in left leg. Cannot bear light; left ptosis. Now, there is complete paralysis of left upper extremity. Muscles at back of neck rigid, with pain on pressure over them. Head turned to the right, left sterno-mastoid rigid. Breathing almost entirely diaphragmatic. Cannot think of words. Marked "tache." Wasting of muscles of left side.

21st.—The only voluntary movements are, putting out the

tongue, opening the eyes and mouth, and moving the eyeballs. Insensitive everywhere except on face.

22nd.—Almost unconscious, cannot swallow, speak, or feel either on skin or conjunctivæ. Regular movements (forty-two per minute) of both eyes round a horizontal axis and through about 48° . Complete paralysis of head, trunk, and extremities, except respiratory movements, the ocular movements just mentioned, and slight movement of left side of face.

23rd.—Quite unconscious, respirations six per minute; very cold, lips livid; died at 4 a.m.

Post-mortem examination.—*Brain*: Convolutions flattened. Left hemisphere bulged outwards and behind, and inwards across the middle line. The convolutions more particularly flattened out were the ascending parietal and ascending frontal together with the posterior extremities of the first, second, and third frontal convolutions. The inferior parietal lobule was more flattened than the superior. The central lobe was unduly buried by the swollen convolutions. The left optic nerve was double the size of the right. When the deeper part of the cerebrum was examined it was found that the cortex was involved to the extent already mentioned, but the white matter to a less extent, so that on horizontal section of the white matter of the centrum ovale a broad band of grey substance occupied that part of it which almost exactly corresponded to the ascending parietal and ascending frontal gyri, being rather more posterior than immediately below them. On making a section at the level of the corpus callosum, the grey change had extended further back into the white fibres supposed to go to the posterior lobes. But it was everywhere noticeable that the grey matter, or rather the white matter turned grey, was wedge-shaped, with the base at the cortex, and that the central ganglia were perfectly healthy with the exception of a minute spot of grey change in the white matter of the external capsule between the lenticular nucleus and the anterior part of the claustrum of the left side. The vessels and the rest of the brain were healthy. Microscopic examination of the diseased part showed clearly that it was gliomatous. The rest of the viscera were healthy.

Spinal Cord much diseased. Where the change had not gone too far to localise it, the grey commissure and the ante-

rior part of the columns of Goll were quite soft and had blood extravasated into them. This changed part squeezed out of the rest of the cord much as oil-paint squeezes out of the cylinders in which it is sold. This central softening began at the space between the sixth and seventh nerves in the cervical region and extended to within an inch of the end of the cord. At the above-mentioned spot in the cervical region was a mass of pulp, which microscopic examination showed to be a very vascular growth; it appeared that the softening had extended down from this. In the pia mater, chiefly on the posterior surface of the cord, were irregular, opaque layers of new growth. These were more especially collected at the points of emergence of the nerves.

CASE 2.¹—J. D—, æt. 32, science teacher, admitted into Philip Ward under Dr. Hilton Fagge, April 21st, 1882.

Family history unimportant.

Personal history.—Has been in comfortable circumstances, has studied hard.

Present illness.—Three years ago violent headache with vomiting: he got quite well of this but soon after he had pain and wasting of the right arm. This improved and, with the exception of headache, he was quite well till January 1st, 1882. He then became stupid and drowsy, would use the wrong words, and at times give a curious laugh, always simultaneously shaking his left leg, closing his left eye, and drawing up the left corner of his mouth. He became very dull and a doctor was called in who gave him iodide of potassium. He began to brighten up a little, but on April 16th he was violently sick, and after this he slept almost continuously for three days. Lately he has always leaned and fallen to the left side, and has been very drowsy and unintelligent, saying absurd and ridiculous things. His doctor says that on January 1st he had ptosis of the left eye and paralysis of the right side of the face.*

On admission.—Temp. 99·0°. Heart and lung signs normal. Left pupil contracted, right normal. Right ptosis. Both optic discs slightly white. Hearing deficient. Forgets the names of things, repeats the last few words of a sentence.

¹ For the temperature in this case, see the chart at the end of the paper.

No anæsthesia. No difference in power of clasp. Reflexes :— Extreme left ankle clonus, slight right. Excessive right plantar reflex. Patellar reflex normal. Epigastric, cremasteric and abdominal reflexes absent.

April 24th.—Very drowsy, headache. Has to be fed.

27th.—Has attacks of stertorous breathing and rattling in the throat. This morning stopped breathing entirely for a few movements. Has now complete paralysis of right third nerve and partial paralysis of muscles of expression on left side ; passes urine and fæces under him.

29th.—Drowsy, mutters to himself. Ankle clonus absent on both sides. Cremasteric, abdominal, and epigastric reflexes obtainable on the right side, but absent on the left.

May 3rd.—Lies comatose except when he is roused to be fed. Can swallow nothing. Passes all urine and fæces under him. Double divergent squint, neither pupil reacts to light ; optic discs the same as before.

8th.—Has been in much the same condition. At 2.30 and 5.30 a.m. had attacks of stertorous breathing accompanied by profuse sweating. Considerable rigidity of limbs.

9th.—Became gradually worse and died to-day.

Post-mortem examination.—*Brain* : Membranes non-adherent and not thickened. Gummatous nodules in the dura mater, one on the posterior margin of the foramen magnum, slightly to the left of the middle line, the other at the anterior extremity of the straight sinus and at its left side ; both these were the size of hazel nuts. There were three non-diffused, yellow, and gummatous masses surrounding the arteries. One was around the anterior communicating and involved both anterior cerebrals, it was three quarters of an inch broad and a quarter of an inch thick, and could be easily raised from the subjacent brain substance which was not involved. It rested on the lamina cinerea and embraced the right optic nerve, which appeared somewhat compressed. The second mass surrounded the middle cerebral of the right side ; it was evidently gummatous and measured about a quarter of an inch in each direction. A third surrounded the right posterior cerebral and was about the same size as the second. There was no absolute impediment to the flow of water through the circle of Willis, but the anterior cerebrals were somewhat obstructed.

There was a patch of softening about the size and shape of an almond in the posterior part of each corpus striatum. Slight broncho-pneumonia due to the sucking in of food. Kidneys pale. Left testis fibroid.

CASE 3.—Wm. E—, æt. 34, admitted into Stephen Ward under Dr. Pavy, October 22nd, 1876.

Family history very good.

Personal history very good. Quite well till six years ago when he attended the Victoria Park Hospital for his chest. Sixteen months ago gradual pain in the head, neck, and spine. This kept him in bed seven months. Has had pain with intermissions till three weeks ago when he was seized with much pain in the head, neck, chest, and arms. Fourteen days ago his left extremities began to feel numb and cold. Ten days ago the same occurred on the right side. Five days ago inability to pass his water.

On admission.—No facial paralysis. Right ninth nerve implicated. *Left arm*: complete loss of movement and impaired sensation. *Right arm*: on admission he could move it; but a few days after admission it became completely powerless, sensation good. *Left leg*: complete paralysis, sensation good, could distinguish between heat and cold. *Right leg*: movement at first good but soon this completely disappeared: thought cold things were hot. *Trunk* healthy, abdominal sensation impaired below nipples. Paralysis of bladder, no incontinence.

24th.—Temp. 100°. Gradually getting worse.

25th.—Morning temp. 102·4°, evening temp. 103°. The temperature of the legs is equal, but in the case of the arms the left is 99·1°, the right 100·8°.

26th.—Morning temp. 101°. Died this evening.

Post-mortem examination.—All the organs were healthy except that there was a sarcomatous tumour attached to the meninges compressing chiefly the upper part of the cervical cord and slightly the medulla. It was not adherent to the cord.

CASE 4.—Charles S—, æt. 27, fireman, admitted under Dr. Pye-Smith into Stephen Ward, August 27th, 1879.

Family and previous history unimportant.

Present illness.—Two years ago he caught cold and had rheumatism with much pain. This was soon followed by weakness of the arms. Nine months ago he became worse and had difficulty in passing his water. His condition from then till now has varied, but he has become much weaker, and for the last month has not been able to walk at all.

On admission.—Weakness of legs, especially right; cannot stand alone. Cannot grasp well with either hand; no facial palsy, no anæsthesia or hyperæsthesia, great pain in the loins and down the spine. Pupils regular, no exaggerated patellar reflex. All the rest of his body is apparently healthy. Temp. 99.2° .

September 1st.—Has been in great pain, morning temp. 99.9° , evening temp. 100.4° .

2nd.—Morning temp. 100.4° , evening temp. 100.4° . After the passing of a catheter this evening he had a severe rigor.

5th.—Patient appears to be sinking fast. 2.30 p.m.—Temp. 105° . 3 p.m.—Temp. 108° . Arms and hands cold and clammy. 3.20.—Temp. 108.4° . 3.35.—Temp. 107.8° . 3.45.—Temp. 107.6° . 4.5.—Temp. 107.6° . Death.

Post-mortem examination.—*Bladder*: Much cystitis. *Spinal cord*: Lying beneath the visceral layer of the arachnoid two and three quarter inches from the lower border of the pons was a dark flat mass two and a half inches long. It occupied chiefly the right lateral half of the cord but extended as far forwards as the median fissure. The posterior roots of the 7th and 8th cervical nerves were considerably stretched over it and also the anterior root of the 7th. The tumour was soft in the interior. The cord was soft for three inches below the tumour. The brain was quite healthy.

CASE 5.¹—Lily M—, admitted under Dr. Bristowe, April 4th, 1882. Two years ago paralysis began in left hand. Later dimness of left eye, and deafness in left ear. A year ago diplopia. Seven months ago left internal strabismus and soon after numbness of the left arm and leg, and some stiffness associated with tremors in the legs. During the last four months the progress has been rapid, she has become deaf and blind, with left external strabismus, she drawls and whines and

¹ Case recorded by Dr. Bristowe, 'Brain,' part xxii, July, 1883.

is incoherent. Her limbs are weak, and all her movements are attended with tremors. On admission the limbs are weak, the head and neck tremble, she is quite deaf and blind, paralysis most complete in right external rectus and weakness of all other ocular muscles on both sides, rotatory nystagmus of the left eye, pupils unequal, double optic neuritis. Amorous in her manner. Slight paralysis of the right facial.

April 18th.—A mattery discharge took place from the right ear; this continued till death.

May 21st.—Up to this date has remained in pretty much the same condition, her temperature not having been important, but to-day it rose unaccountably to 102.2° .

22nd.—Temp. 103.2° , more drowsy.

25th.—Began this evening to tremble violently in the arms, legs, trunk, and head and neck. Temperature between 101° and 102° .

29th.—Could not stand, choked a good deal on taking food. Veins of forehead and inner extremities of the eyelids much distended; from this time she gradually sank and died on the morning of the 30th. Her temperature, which during the last ten days had varied a good deal, but on the whole had remained high, rose an hour before death to 105.4° .

Post-mortem examination.—Slight general congestion of the surface of the brain with flattening of the convolutions; the lateral ventricles were largely distended with fluid. Originating in the valve of Vieussens was a soft translucent vascular new growth which extended into the neighbouring part of the cerebellum nearly to its middle lobe, and appeared to involve to some extent the corpora quadrigemina, which looked swollen and cedematous. It was adherent to the floor of the 4th ventricle, the ependyma of which was converted into a thick pulpy layer, but it did not appear to have encroached upon the subjacent medulla. The growth was the size of a bantam fowl's egg; the limits between it and the nervous tissue were very ill-defined. The rest of the brain and also the cord were healthy. Patches of dura mater in both anterior fossæ, in both middle fossæ and in a less degree in both posterior fossæ, were the seats of growths identical with that affecting the brain. They formed irregular tracts, a line or two thick, cent, slightly adherent to the surface of the brain

above, and caused erosion and honey-combing of the subjacent bone tissue. Some of the nerves issuing from the skull seemed to have been pressed on by these growths; and it was believed that the affection of the ear observed during life was due to the affection of the dura mater. Other viscera healthy.

CASE 6.¹—Henry A—, æt. 3, admitted into the Victoria Hospital for Children under Dr. Ridge Jones, May 10th, 1883. Two years before admission patient had measles, since then his head has been growing large. For a year before admission has squinted, has had fits, loss of power and contraction of the right arm, head turned to right side, palsy of left face with impaired vocalisation.

On admission.—All the above symptoms were present with optic neuritis and involuntary passage of urine and fæces.

May 23rd.—Temperature rose all day, child drowsy, right extremities extended and stiff, difficulty in swallowing, tongue and mouth both drawn to left side.

24th.—Semi-comatose. Temperature 108°, skin dry and burning hot.

25th.—Face blanched, with a temperature of 105°. Child died at 8.30 a.m.

Post-mortem examination.—Two small excavations on the cranial surface of each crus cerebri. Pons varolii more prominent on the left side than the right. On section a yellowish circumscribed mass was seen pressing on the floor of the fourth ventricle. Microscopic examination showed the tumour to be a tubercular mass. Every other part of the body was healthy.

CASE 7.—F. D—, a boy, æt. 11, admitted into the National Hospital for the Paralysed and Epileptic under Dr. Ramskill, June 2nd, 1880. Present illness came on five months ago. Four months ago he had a fit, afterwards paralysis of the muscles of the eyes, left arm and leg, and left face.

On admission.—All the above parts were paralysed. There was intense double optic neuritis. He was deaf. He passed

¹ Ridge Jones, 'Brit. Med. Journ.,' vol. ii, 1883, p. 821. For the temperature in this case, see the chart at the end of the paper.

urine and fæces under him. Persistent priapism. Constant vomiting. Temperature the evening before death 104° ; just before death 107.4° .

Post-mortem examination.—A psammoma three inches long and two and a half broad growing from the upper surface of the cerebellum, extending forward between the optic thalami, flattening the corpora quadrigemina, pressing upon the right crus, and the right superior cerebellar peduncle.

CASE 8.¹—This case is reported at full length in the previous volume of these reports, and therefore it will suffice to say that the patient was a boy, W. C—, æt. $6\frac{1}{2}$, who was admitted into the Evelina Hospital under Dr. Frederick Taylor. His chief symptoms before admission were paralysis of the right side, which came on suddenly and was followed by incontinence of urine. On admission three months after this, he was found to be weak on the right side, he never spoke, took no notice of things going on around him, and passed all his urine and fæces under him. For a month his temperature remained normal or subnormal, and his condition remained unchanged, but after that time had elapsed the temperature rose to 102.4° , and the boy had a fit at the same time. From this time to his death it frequently rose very high, sometimes the rise was accompanied by convulsions, sometimes it was not. About three weeks before his death he became completely unconscious and remained so, he refused all food by the mouth, and had to be fed with enemata, and his limbs became rigid. Whenever the temperature rose above 103° the child was sponged, but, as will be seen from the account of the case in the last volume of these reports, it was even by this means impossible to keep it down as it rose so rapidly, often reaching 106° or even 107° . His death occurred three months after admission.

Post-mortem examination.—Rigor mortis well marked. Brain remarkably firm, vessels and vascular spaces larger than normal. No evidence of tubercle, meningitis, tumour or disease of the cerebral arteries. Near the anterior extremity of each corpus striatum, just above the anterior commissure, was a brownish patch, about a quarter of an inch in diameter,

¹ 'Guy's Hospital Reports,' vol. xli, 1882, p. 21.

so soft that it left a hole when the brain was cut ; these patches were symmetrical, both in colour, size, and position. Well marked grey discolouration was present in both crura cerebri at the anterior inner margin, this tract of degeneration was also very distinct in both crossed pyramidal tracts especially the left, which could be traced up to a communication with the degenerated tract in the left brain. This degeneration of the cord extended down to the lumbar region. There was also some degeneration in the cord on the inner margin of the left anterior column. All the other organs in the body were perfectly healthy.

CASE 9.—B—, æt. 40, admitted into the Philadelphia Hospital May 20, 1879. Three weeks before an attack of vertigo, after which he noticed that his speech was thick, and that the right arm and leg were paralysed.

On admission.—The only important symptoms were right hemiplegia and left convergent strabismus. On the day after admission sudden paralysis of left arm. Three days later a peculiar seizure, with difficult respiration, profuse perspiration, foaming at the mouth, inability to speak, helpless in all his limbs, difficulty of swallowing. His condition remained unchanged for three days, his temperature being about 100°. On the fourth day much more profuse sweating and involuntary discharges. His temperature rose till five hours before death it was 108°.

Post-mortem examination.—The pons was the seat of an irregular area of softening, the centre of which was just below the centre of the pons.

CASE 10.—James P—, æt. 65, admitted under Dr. Hilton Fagge in Clinical Ward, May 9th, 1878. Whilst digging he was suddenly attacked with giddiness. He was helped by his comrades, became gradually insensible, and was brought to hospital.

On admission.—Semi-conscious, aphasic, right hemiplegia. Temp. 100·2°. Stertorous breathing.

May 10th.—Still unconscious, passes urine and fæces under him. Temp. 100·2°.

11th.—More drowsy, cannot protrude his tongue. Temp.

102·8° in the afternoon; towards evening the temperature rose rapidly and he died at 9 p.m.

Post-mortem examination.—Brain 55 oz. Arteries thick, rigid, yellow. Left hemisphere ploughed up by black, recently clotted blood. The following parts were free:—occipital lobe, frontal lobe, corpus striatum, optic thalamus, capsules, ventricles, and Broca's convolution. Under the pia mater of the cerebellum, chiefly on the upper but also on the lower surface, was a hæmorrhagic effusion evidently recent, which also extended slightly into the grey substance. At the front of the right corpus striatum was a small old hæmorrhage. Heart much hypertrophied, kidneys granular.

CASE 11.—Michael F—, æt. 40, a navvy, admitted into Clinical Ward under Dr. Habershon, October 27th, 1876. Has drunk much.

Family and personal history.—Unimportant. Was quite well till 4 p.m. to-day, when he felt a cramp in the arm and was caught in the act of falling. He was taken to a public-house and had some brandy given him. There was neither loss of consciousness nor convulsion.

On admission.—Is perfectly conscious, speech thick, face congested, pupils minutely and equally contracted. There is loss of sensation and motion in the left arm, and he is gradually losing power in the left leg. Partial loss of sensation, left thorax, and abdomen. Slight left facial paralysis. Heart, lungs, and urine normal. Temp. left axilla 95·6°.

7.20 p.m.—Vomited.

11 p.m.—Vomited, insensible. Left pupil smaller than right.

October 28th.—10.30 a.m. Completely unconscious, both extremities and both sides of the face paralysed and anæsthetic. Skin hot and perspiring. Temp. right axilla 105·4°, left axilla 106·8°; pulse 136.

12 noon.—Is sinking fast, the contracted pupil is gradually dilating. Temp. right axilla 107·2°, left axilla 107·4°.

12.15 p.m.—Died. Temp. both axillæ 107·2°.

12.30 p.m.—Temp. 100·4°.

8 p.m.—*Post-mortem examination.*—Rigor mortis well marked. Arachnoid cavity distended with serum. Arteries

tortuous. Brain weighed 52 oz., both sides equally flattened. Corpus callosum arched. Left ventricle widely distended with blood-stained fluid. Right ventricle walls soft, distended with clot and fluid, laceration between the corpus callosum and optic thalamus. Septum lucidum torn through. Third ventricle filled with blood-stained fluid. Fourth ventricle contained a diamond-shaped clot. Right insula flattened. Some ecchymosis around the clot in the brain. Kidneys: left hypertrophied, 9 oz.; right, cortex atrophied. Heart hypertrophied, 15 oz.

CASE 12.—Michael B—, æt. 43, admitted into Clinical Ward, under Dr. Pye-Smith, March 11th, 1883. Has had gout, no syphilis; has drunk freely. Had an attack of faintness and loss of vision a week ago. Was found this morning unconscious, sitting on some stairs, with his legs drawn up.

On admission—Midday.—Breath foul; unconscious; pulse rapid; arms and jaw rigid; pupils small and insensible to light; eyes directed to the left; rigidity of shoulders, elbows, and hips; knee-jerk exaggerated; no ankle clonus; circulatory and respiratory systems normal; urine slightly albuminous; temperature 98·6°.

March 12th.—Still unconscious. Morning temperature 100°. 4 p.m.—Temp. 100°. Died 11 p.m.

Post-mortem examination.—The only point of interest bearing on the question of heat-fibres was, that there was a hæmorrhage of the size of a large walnut, just outside the right lenticular nucleus.

CASE 13.—Henry W—, admitted into Accident Ward under Mr. Davies-Colley, December 20th, 1883, having shot himself with a pistol.

On admission.—Insensible, small triangular wound on the right side, between the external angular process and the top of the pinna, with depressed bone at the bottom; right orbit distended and eye protruded; eyelids black from extravasation; respiration irregular, 28—35; pulse 52, strong, regular, full; right pupil widely dilated, left strongly contracted, both insensible to light; face not distorted. Almost directly after admission the bullet was extracted; it was found to be at the

apex of the orbit. During the operation he frequently moved both arms. He never recovered consciousness; Cheyne-Stokes respiration became very marked, and he gradually sank and died about twelve hours after the operation. Immediately after the operation the temperature was 99.2° , but it ran up in the course of four hours to 104.2° . It remained high, being half an hour before death 104.4° .

Post-mortem examination.—Several pieces of bone were found broken, especially the lesser wing of the sphenoid. The bullet had not perforated the dura mater. The anterior extremity of the middle lobe of the right hemisphere was severely contused, as was also the third right frontal convolution; the damage affected the whole thickness of the grey matter. Hæmorrhage in the arachnoid in this situation.

CASE 14.—Wm. G—, æt. 48, admitted into University College Hospital, February 9th, 1877. Rheumatic history; admitted for dyspnœa and albuminuria. Ten days after admission had a severe attack of dyspnœa without apparent cause; his pupils became contracted. The next morning he lay on his back breathing stertorously; skin hot, perspiring; pupils contracted, equal, insensible to light; could hardly be roused; limbs rigid.

At 3 a.m. the next day the coma was deeper, swallowing impossible, temperature 105.6° ; at 5 a.m. it was 109.2° . A quarter of an hour after this he died.

Post-mortem examination.—Heart dilated; weighed 23 oz. Kidneys granular. The anterior half of the basilar artery contained a clot, the anterior part of which was evidently an embolus. The superior cerebellar and posterior cerebral arteries both contained fluid blood.

CASE 15.¹—Wm. G—, æt. 49, admitted into Philip Ward under Dr. Moxon, April 11th, 1881, for locomotor ataxy. Both his arms and legs were affected; he had the Argyll-Robertson symptom. The larynx showed very little movement of the vocal cords which were close together and only moved slightly during inspiration. Voice high pitched, whistling. Has a difficulty in uttering low notes, and when spoken

¹ A temperature chart of this case will be found at the end of the paper.

to prepares himself for answering by drawing a long breath ; this action is accompanied by stridor. Diplopia and ptosis.

June 18th.—Last night temp. 103.2° and he was very feverish ; complains that his feet are cold and they certainly feel colder than the rest of the body.

20th.—By this morning the temperature has sunk to normal and the feverish attack has passed off.

25th.—After being normal or slightly subnormal the temperature rose this morning to 102.4° ; was in much pain all night.

29th.—There is no further record of the temperature till to-day when it is noted that he had a fit of shivering, was very flushed, and at 2.30 p.m. his temperature was 103.2° , pulse 102, resp. 42 ; cramps in the stomach and pains in various parts of the body ; the muscles of the abdomen are contracted.

July 6th.—The patient's attack has gradually passed off and now his temperature is normal ; it has been ranging slightly above normal the last few days.

CASE 16.¹—Charles B—, æt. 22, is a police constable, admitted into Clinical Ward, April 14th, 1876, under Dr. Frederick Taylor.

Family history.—Good.

Personal history good except that when five years of age he is said to have had sunstroke from which he recovered in a week.

Present illness.—At 6 p.m. yesterday whilst writing at a desk his hand shot straight out, his face became distorted, but he did not lose consciousness ; he had his tea and again went on with his work. About 7 p.m. he was heard to fall, was picked up insensible and foaming at the mouth ; the saliva was a little bloody. He was throwing his limbs about and was difficult to hold. Taken into the fresh air he vomited and recovered. The fit lasted about ten minutes and when it was over he walked back to the mess-room. Twenty minutes later, during another fit, he would have fallen but was caught. Face was not distorted during either of the last two attacks.

On admission (2 p.m).—Strong-looking man, lying on his back apparently quite unconscious, in which condition he had been for at least twelve hours previously ; skin hot and moist,

¹ For the temperature of this case, see the chart at the end of the paper.

no paralysis of sensation or motion. Pupils dilated, equal. Temp. 100.2° , pulse 96, regular, resp. 36. Heart, lungs, and urine, all apparently normal.

April 16th.—Has been unconscious till this morning, but now he is quite conscious. Can give no history of his attack.

17th.—At 6.15 a.m., whilst nurse was getting him out of bed, he began shivering; arms and legs became rigid, and quivering; was very pale, but the face did not work; they got him back to bed and he was found to have become suddenly unconscious with stertorous breathing.

11 a.m.—Whilst being examined the patient became suddenly livid and respiration stopped for about four minutes. The heart's action continued strong but irregular. Artificial respiration brought him round.

2 p.m.—During the last three hours has frequently required artificial respiration, pupils constantly dilating and contracting, nystagmus. Skin hot.

10 a.m.—Has required no more artificial respiration and is now becoming conscious.

18th.—Appears fairly sensible, pupils normal.

19th.—Is improving, answers some questions, but soon rambles.

21st.—At 5.30 a.m., had an epileptiform attack, in which he was unconscious for half an hour.

11 a.m.—Is now unconscious, again very livid, general tremors, shaking the bed, eyes in constant motion, pupils irregularly dilated, limbs flaccid, profuse perspirations, respirations frequently suspended for thirty seconds. Temperature running up. All these symptoms increased and he died at 1.40 p.m.

Post-mortem examination.—Except for some congestion of the lungs and kidneys, the whole of the organs of the body were perfectly healthy. The brain was most minutely examined.

CASE 17.—Eleanor B—, æt. 25, admitted into Mary Ward, under Dr. Wilks, February 18th, 1878.

Family history good, was admitted for ovarian pain following the birth of her child nine months ago.

On admission.—Skin hot and moist, menstruation irregular,

complete anæsthesia of left side of body, no hæmorrhage when pricked, some deafness in left ear and colour blindness left eye. Temp. 99° . Otherwise well.

February 20th.—Temp. 100° . Sweating profusely.

21st.—Gold metallo-therapy was tried on the anæsthetic side; it slightly improved the sensation on that side, but diminished it on the other; after this treatment the blood flowed on pricking.

22nd.—Has had a bad night with rigors and intense headache, sweating profusely, face flushed.

26th.—The condition mentioned in the last note has continued till now when gold metallo-therapy was again applied. After this she slowly improved in all her symptoms, metallo-therapy being frequently applied. She left the hospital much better than when she entered it.

CASE 18.—Jane P—, admitted under Dr. Wilks, September 18th, 1879. Had been previously in the hospital twice for hysteria, once under Dr. Pavy and once under Dr. Goodhart; the last time her chief symptoms were sinking pain in the epigastrium, attacks of sobbing, &c.

On admission.—Looks healthy, the only abnormalities that could be detected were that the skin was hot and dry, and there were a few rhonchi in the lungs. She says that the touch of the stethoscope gives her pain. Temperature 102.2° .

September 9th.—Temp. 98.8° .

20th.—Temp. 102° .

21st.—Morning temp. 103° , skin hot; from this date her temperature remained up for several days, but then fell to normal; ultimately she was discharged quite well.

CASE 19.—Rosa B—, æt. 33, admitted into Mary Ward, under Dr. Frederick Taylor, August 30th, 1883, and afterwards under Dr. Pavy.

Family and personal history unimportant. No history of malaria. Six weeks before admission had a shivering fit accompanied by a rash which spread upwards from the legs; has not been well from that time till admission, having great pain in the legs, loss of appetite, and scanty urine.

On admission the only notable points were that there was

a papular rash present, and the urine was high coloured, contained many urates and one-third albumen. The report of the patient's condition during her stay in the hospital is too long to repeat here. Her chief symptom was violent rigors without any assignable cause for them being discoverable. During the whole of her stay in the hospital there was hardly a day during which the temperature, at one period of the twenty-four hours, was not normal or thereabouts, and at some other period considerably raised. These rises were curiously regular. Thus for a week the highest point, which was about 103° , would always be reached at noon, then for a series of days at 3 p.m., then it would again vary to some other time. She wasted considerably and ultimately had some œdema about the ankles. For a long while she was subject to a papular rash. Towards the end she became a peculiar yellowish colour. The pains in the legs troubled her a great deal. At times the spleen was enlarged and the liver slightly so. No lung symptoms developed till towards the end when she got crepitations at the bases. The condition of the urine was variable, the albumen disappeared. Until September 17th she had diarrhœa. The pelvic organs were healthy. She gradually sank and died on November 15th without any cause whatever being found for the rigors and hyperpyrexia.

The temperature chart on Plate II at the end of this paper gives the highest and lowest record daily for the first month after admission; and the pyrexia was similar till the day of her death. The hours recorded under the dates of the month give the times at which the highest daily temperature occurred.

Abstract of post-mortem report.—Cystic kidneys; acute right pleurisy; general acute bronchitis; no pus in shoulders, elbows, wrists, hips, knees or ankles; no clotting in femoral vein; heart healthy; brain and cord healthy; bones of skull and internal ear healthy; piece of tibia removed and found healthy; liver fatty; gall-bladder contained small quantity of bile; spleen large and of natural firmness; right suprarenal body somewhat swollen; no cause of the pyrexia was ascertained.

For more ready reference the cases in which a nervous lesion was found post-mortem have been tabulated.

Cases with Lesion and Post-mortem Examination.

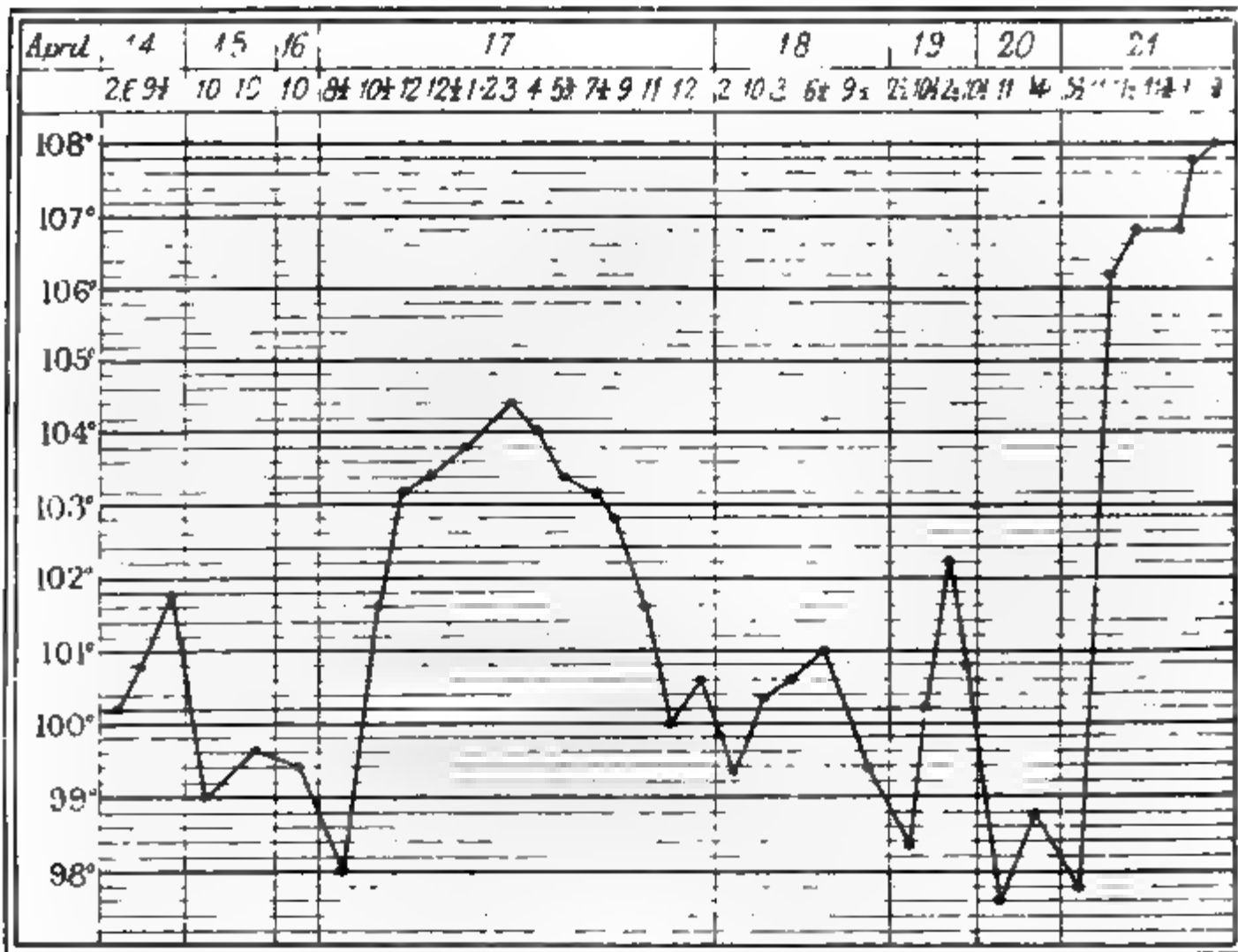
A.—Tumours.

No. of case.	Under.	Remarks.
I	Dr. Pye-Smith	viscera healthy.
II	Dr. Hille Fagge	stis fibroid.
III	Dr. Pav	n the hospital a ays; other viscera hy.
IV	Dr. Pye-Smith	<p>On admission temp. 99° F; it rose during the whole of the illness, so that shortly before death it was 103° 4°</p> <p>Beneath the visceral layer of the arachnoid, 2½ inches from the lower border of the pons, is a tumour 2½ inches long, occupying the right lateral half of the cord. The posterior roots of seventh and eighth cervical nerves were stretched. The cord was soft for 3 inches below the tumour</p> <p>Meach cystitis; brain healthy.</p>

XI	Dr. Habershon	death Temp. fell to 95.6°; before death rose to 107.4°	optic thalamus, capsules, ventricles, and Broca's convolution. Clot under the pia mater on the upper surface of the cerebellum Very extensive hemorrhage filling all the ventricles	Recorded because of the unusual range of temperature, viz. 11.8°. Fractured his skull by the fall which occurred when the apoplexy came on.
XII	Dr. Pye-Smith	Temp. never sank, but for some hours before death it was 100°	A hemorrhage, the size of a large walnut, just outside the right lenticular nucleus	
XIII	Mr. Davies-Colley	Operated on admission. Immediately after operation the temp. was about 99°, but it rapidly rose till it reached 104°, where it remained till death	Several pieces of the lesser wing of the sphenoid broken. The anterior extremity of the middle lobe of the right contused, as was also the third. The damage affected the whole	Nothing else to account for the rise of temperature.
XIV	Dr. Gowers (recorded in 'Brain')	It ran up very high, so that at death it was 109.2°	The attack came on somewhat suddenly, with stertorous breathing, insensibility, and contracted pupils	Dilated heart, granular kidneys.

D.—Injuries to Brain.

E.—Embolism.



LIST OF SPECIMENS ADDED TO THE PATHOLOGICAL MUSEUM

DURING THE YEAR 1882-83.

By JAMES F. GOODHART, M.D.

I HAVE often thought that it would be a good thing if the Curators of the museums attached to the large metropolitan hospitals, following the example first set by Professor Flower at the Hunterian Museum of the Royal College of Surgeons, were, at some appointed time during the year, to have an exhibition of all the specimens added to the shelves within the previous twelve months. It might perhaps be said by a superficial critic that what is a good thing for a national museum is not necessarily advisable for more private or local enterprise, but to such an one it will be sufficient to reply that the museum of any large hospital is a part, and a very important part, of the records of medicine. It is needless to say that its supplies are of the richest; all the more rare conditions are kept on the spot, and the Hunterian Museum—unfortunately, as all must admit—in very many respects, even at its best, comes off but poorly. None, therefore, of the museums of the medical schools that have been at all well kept up are places of mere local concern, and that of Guy's Hospital is admitted by all to be pre-eminently rich in specimens. On this account therefore, as a matter of public interest and concern, the Curator should show its treasures. But I do not stop there: all of us know well that year by year it

becomes increasingly difficult to procure adequate opportunities at the various societies for the exhibition of the many interesting specimens that collect all too quickly. With numbers crowding into the profession, all eager for work, few without something to show and to say, I venture to think that there is not a demonstrator of morbid anatomy in the metropolis who has not a number of interesting and instructive specimens which have never seen the light. They might not see much light with an annual day of exhibition, but they would at any rate have their chance, and it is only this that I would ask for them.

There are other more homely uses of such an exhibition ; the museum becomes then a more active centre of interest and work. Museum officials, curators, sub-curators, assistants, voluntary workers, like the world outside, work all the better the more the interest that is shown in their work. And donors too, seeing their gifts well tended, well displayed, new methods of mounting, and so on, better appreciate what a museum wants, in what condition specimens should arrive there, and the lessons a good preparation can teach. They are thus far more likely to interest themselves to secure valuable preparations, and to send them in a state in which they are likely to prove serviceable.

Museum shelves must have many a curious tale to tell of how and why they acquired their furniture. This specimen because Mr. So-and-so cut it off ; this because Mr. A. or B. gave it ; this because, though a very ordinary thing, it came from some extraordinary man ; this to record the fact that in it there is nothing to show ; this apparently that what there was to show has long ceased to be appreciable ; this because it is published somewhere, and so on. I might even give more remarkable reasons for the putting up of preparations than this. But let these give a general indication of what should be the principle of selection by their want of any principle ; and let them also convey that in the best interests of any museum a Curator has often, very often, to run counter to estimates born of enthusiasm, in other words, to say that this and that are not worth mounting ; and that donors (if they do not forget all about their gifts) should cultivate that charity which forgiveth all things.

Lastly, an annual exhibition necessitates the preparation of some such list of the specimens as that which follows, and it

has been thought by my colleagues and myself that if this be published year by year in the 'Guy's Hospital Reports' it will not only call attention to preparations which might otherwise be forgotten by those in whose practice they have occurred, but it will serve as a useful memorandum for reference and addition to the private catalogue.

The labour of preparing and mounting these numerous specimens has, naturally, fallen upon our valued Museum Superintendent, Mr. Betts, and his Assistant, Mr. Hunt. This, the adoption of improved methods of mounting and the re-mounting of a large number of old specimens, constitute, in combination with other somewhat multifarious duties, a year's work which we shall all feel they can contemplate with satisfaction.

1000²¹. Caseous Ostitis of Humerus, with Caseous Consolidation of Lung. *Mr. Howse.*

1000²². Syphilitic Disease of Humerus in an Infant.

Dr. Goodhart.

1000^{23 to 27}. A Series of Five Preparations showing a remarkable Softening and Overgrowth of the Bony Skeleton of an Infant.

Dr. Hilton Fagge.

1000²⁸. The Calvaria of an Infant, aged two years, with M. Parrot's Nodes upon the Surface. *Mr. John Poland.*

1000²⁹. The Calvaria of an Infant, aged six months, with Membranous Opercula replacing the Bone in all directions.

Mr. Bryant.

1073²⁰. Osseous Tumour of Frontal Bone growing into the Brain.

Mr. Jacobson.

1073²⁶. Sarcomatous Tumour of Temporal Region.

Mr. Bryant.

1089¹⁰. Fracture of Nasal, Frontal, and Ethmoid Bones.

Mr. Howse.

1117⁴¹. Old Colles's Fracture.

Mr. Davies-Colley.

1134⁸⁰. The Femur of Leucocythæmia with excess of Red Marrow.

Dr. Goodhart.

1155³⁰. Abscess in the Great Trochanter.

Mr. Howse.

- 1211⁸³. Vertical Fracture of Patella united by Bone.
Dr. Goodhart.
- 1315⁵. Healed Ulcers in the Cartilage of the Head of the Femur.
Dr. Wilks.
- 1315⁵⁵. Acute Disorganisation of the Hip in an Infant eight months old.
Mr Davies-Colley.
- 1318⁸³. Femur upon which Osteotomy had been performed six weeks before death.
Mr. Bryant.
- 1318⁶⁰. Ankylosis of Vertebrae and Hip. *Dr. Hilton Fagge.*
- 1329⁵⁶. Abscess opening into the Knee-joint. *Mr. Bryant.*
- 1364²⁶. Bursa Patellæ, with Loose Cartilage within it.
Dr. Carrington.
- 1394²⁶. Acute Dilatation of the Left Ventricle during Scarlatinal Nephritis.
Dr. Goodhart.
- 1395³¹. Cured Aneurism of the Heart. *Dr. Habershon.*
- 1399⁶⁶. Secondary Cancer of the Heart Muscle.
Dr. Braxton Hicks.
- 1400⁴¹. Laceration of Right Auricle. *Dr. Goodhart.*
- 1400⁷⁶. Acute Fatty Degeneration and Myocarditis from Disease and Thrombosis of the Coronary Arteries. Rupture of the Left Ventricle.
Dr Moxon.
- 1401²⁶. Ulcerative Endocarditis of Tricuspid Valve with Perforation of the Septum. *Dr. Moxon.*
- 1450⁴⁷. Dilatation of the Pulmonary Artery and Right Heart.
Dr. Moxon.
- 1480¹¹. Aneurism of Intra-pericardial portion of Arch of Aorta.
Dr. Pye-Smith.
- 1480⁵⁵. Aneurism of the Arch of the Aorta, opening into the Pulmonary Artery. *Dr. Goodhart.*
- 1489⁵¹. Aortic Aneurism opening into the Lung.
Dr. Goodhart.
- 1494⁷⁶. Abdominal Aneurism rupturing into the Mesentery.
Mr. Bryant.
- 1564⁴¹. Gliomatous Enlargement of the Pons Varolii.
Dr. Goodhart.
- 1564⁵⁶. A Gruyère Cheese Brain. *Dr. Hale White.*
- 1564⁵⁸. The Brain from a case of General Paralysis.
Dr. Mackenzie Bacon.
- 1565⁹¹. Abscess of Brain from Sloughing Naso-pharyngeal Polypus.
Mr. Bryant.

- 1565⁹². Cerebral Abscess. *Mr. Laidlaw Purves.*
- 1575¹¹. Old Apoplectic Cyst of Large Size, outside the Extra-Ventricular Nucleus of the Right Corpus Striatum. *Dr. Hilton Fagge.*
- 1580⁷⁶. Localised Softening in the right third frontal Convolution. *Dr Pavy.*
- 1583²⁶. Yellow Tubercle of the Cerebellum. *Dr. Hale White.*
- 1583³⁷. Yellow Tubercle of the Pons Varolii. *Dr. Goodhart.*
- 1590¹². Dilatation of the Cerebral Ventricles, from a Large Cheesy Tubercle in the Cerebellum. *Dr. Goodhart.*
- 1590^{13, 14}. Two Preparations showing the Extreme Amount of Dilatation of the Fourth Ventricle, which sometimes occurs in Chronic Hydrocephalus. *Dr. Goodhart.*
- 1590¹⁵. A Section of the Cerebellum and Pons Varolii and Medulla, from a case of Chronic Hydrocephalus in which the Fourth Ventricle is not dilated. *Dr. Moxon.*
- 1593⁶¹. Fracture of the Skull, with Rupture of the Meningeal Artery and Extravasation of Blood upon the Surface of the Dura Mater. *Dr. Goodhart.*
- 1602³³. Fibrous Tumour of the Dura Mater. *Mr. Martin.*
- 1652¹. Hair from a Case of Plica Polonica. *Dr. R. M. Simon.*
- 1652⁷¹. Congenital Myxo-lipomatous Tumour from the Thigh. *Mr. Howse.*
- 1657¹⁰. Lympho-sarcomatous Ulceration of Buttock. *Mr. Davies-Colley.*
- 1690⁵. Extreme Tubercular Disease of the Mucous Membrane of Larynx. *Dr. Pary.*
- 1697⁶. Syphilitic Ulceration and Stenosis of Larynx. *Mr. Lucas.*
- 1697²¹. Membranous Inflammation of the Epiglottis due to a Scald. *Mr. Davies-Colley.*
- 1697²². Tracheitis due to the Impaction of a Piece of Nutshell. *Mr. Jacobson.*
1699. Lupus of the Larynx and Fauces. *Mr. Bryant.*
- 1711⁶⁷. Cancer of Thyroid surrounding the Trachea. *Dr. Mahomed.*
- 1711⁹³. A Larynx and Trachea invaded by Cancer of the Thyroid. *Dr. Taylor.*
- 1713⁶⁸. Syphilitic Ulceration of the Bronchus and Lung. *Drs. Miller and Pye-Smith.*

106 *List of Specimens added to the Pathological*

- 1729⁶⁰. Chronic Disease of the Base of the Lung, with Tubercle
at the Apex. *Mr Durham.*
- 1737¹⁶. The Lung of a Child studded with Miliary Tubercle.
Dr. Goodhart.
- 1743²⁹. Gumma in Lung. *Dr. Moxon.*
- 1750³⁸. Colloid Cancer of the Lung. *Dr. Pye-Smith.*
- 1751³². Cancer of the Lung, ? originating in a Vomica.
Dr. Pavy.
- 1769¹. Tuberculosis of Lung and Pleura, with Emphysema.
Dr. Pavy.
- 1780⁶⁵. Cancer of the Pleura. *Dr. Wilks.*
- 1780⁸¹. Ulceration of Caseous Glands into Trachea.
Dr. Pavy.
- 1784¹³. Syphilitic Disease of Diaphragm and Capsule of the
Spleen. *Mr. Cooper Forster.*
- 1784⁶⁸. Epithelioma of Pharynx. *Mr. Durham.*
- 1789⁴⁶. Myoma of the Œsophagus. *Dr. Pavy.*
- 1792²⁰. Cancer of the Œsophagus, Trachea, and Lung.
Dr. Moxon.
- 1794⁵⁰. An Unusual Form of Mammillation of Stomach.
Dr. Wilks.
- 1798⁵. Gastric Anthrax. *Dr. Mahomed.*
- 1813³⁴. Colloid Cancer of the Stomach. *Dr. Pye-Smith.*
- 1831²¹. Acute Ulcerative Enteritis above a Hernia.
Mr. Jacobson.
- 1845^{23, 24, 25, 26}. A Series of Preparations of Lympho-sarcoma
of the Bowel, all from the same Patient.
Dr. Goodhart.
- 1849¹⁷. Chronic Intussusception. *Dr. Mahomed.*
- 1860⁴¹. Hypertrophy, Dilatation, and Ulceration of Colon.
Dr. Goodhart.
- 1864³². Acute Ulcerative Colitis from a case of Colloid Cancer
of the Stomach. *Dr. Pye-Smith.*
- 1864^{51, 52}. Acute Entero-Colitis in Bright's Disease.
Dr. Hilton Fagge.
- 1884²¹. Section of the Female Pelvic Viscera with Chronic
Ulceration and Stricture of the Rectum. *Mr. Bryant.*
- 1884³¹. Simple Ulceration of the Rectum. *Dr. Taylor.*
- 1886¹⁶. Cancer of the Rectum removed by the operation of
Colectomy. *Mr. Bryant.*

- 1886⁴⁹. Cancer of the Rectum. *Dr. Hilton Fagge.*
- 1886⁷⁶. Section of the Male Pelvic Viscera, with Colloid Cancer of the Rectum and Stricture, the disease extending to the Bladder. *Mr. Howse.*
- 1888⁹¹. Sigmoid-vesical Fistula resulting from Latent Dysentery. *Mr. Bryant.*
- 1906²⁶. Cirrhosis of Liver in a Child. *Dr. Wilks.*
- 1906⁹². Thrombosis of Portal Vein from Cirrhosis. *Dr. Taylor.*
1935. Sarcoma of the Liver. *Dr. Fagge.*
- 1955²⁶. A Warty Gall-Bladder. *Dr. Pavy.*
- 1959⁶¹. Rupture of the Gall-Bladder from Injury. *Mr. Jacobson.*
- 2020³³. The Parts concerned in Addison's Disease displayed by dissection to show the relation of the diseased Capsules to the Abdominal Sympathetic, and the Condition of the larger Trunks and Ganglia. *Dr. Goodhart.*
- 2062⁶¹. A Small Calculus with Blood as a Nucleus, passed with others from the subject of Malignant Disease of the Kidney. *Dr. Owen Rees.*
- 2104⁷⁹. Fistula between the Bladder and Rectum Seven Years after Lithotomy. *Mr. Davies-Colley.*
- 2104⁹⁴. Perforation of Bladder by Catheter. *Dr. Goodhart.*
2245. Papilloma of both Ovaries. *Mr. Durham.*
- 2384⁶¹. Hæmatocele. *Mr. Lucas.*
- 2486²¹. Inguinal Hernia reduced *en masse*. *Mr. Lucas.*
- 2502⁶¹. A Knuckle of Bowel from the Sac of a Femoral Hernia. *Mr. Golding-Bird.*

CERVICAL AND BICIPITAL RIBS IN MAN.

By ARBUTHNOT LANE, M.S.

FIG. 1 shows a vertical section through the sternum and costal cartilages of a subject from the post-mortem room of Guy's

FIG. 1.

Hospital. I was unable to make an examination of the rest of the thoracic wall, so that the description is not so complete as I should have wished.

The lower margin of the presternum is directed obliquely

from above downwards and to the left, its edge being irregular. The interval between the first and second pieces of the sternum is very narrow, and is filled up by dense fibrous tissue, presenting on vertical section no indication of a synovial space..

Owing to the obliquity of this articulation, the left second, third, fourth, fifth, and sixth costal cartilages are attached to the sternum at a much lower level than the right. Neither of the seventh costal cartilages reaches the sternum, but each is united by fibrous tissue to the cartilage of the sixth rib.

Between the fourth left costal cartilage and the sternum is a complete arthrodial joint, but with the exception of partial synovial spaces between the second, third, and fourth right costal cartilages and the mesosternum, the articulations consist of a fibrillation of the cartilage without the formation of any synovial membrane in it.

The first costal cartilages are much broader than usual at their attachment to the sternum. The right cartilage is at this point one inch broad, the left is one inch and a quarter. The right increases in breadth as it extends outwards, reaching a maximum of one inch and a quarter at its bifurcation. An inch from its inner extremity it divides into two separate cartilages. The lower is five eighths of an inch in breadth, one inch and a quarter in length. It is continued by a rib, which with its cartilage, resembles the normal second rib and cartilage in shape, but takes the general direction of the first.

The upper branch of the bifurcation is five eighths of an inch long, and half an inch broad, and its line of junction with the rib that continues it is sinuous. It has small patches of osseous formation scattered through it.

The rib joining it is five eighths of an inch broad, and has been divided three quarters of an inch from its inner extremity. It is equally broad throughout, resembling the rib just below it in appearance, but it is more obliquely placed. The interval between these two ribs and cartilages is occupied by intercostal muscles, both internal and external.

The first costal cartilage on the left side is cut at a distance of one inch and three-quarters from the sternum, measured along its lower convex margin. At about half an inch from the margin of the presternum the cartilage is perforated midway between its upper and lower borders by an oval foramen, which

is about half an inch broad. Above this foramen the cartilage presents in its substance a round bony deposit, while outside the foramen is seen the inner extremity of a broad rib, probably the united first thoracic and cervical ribs. The vertical measurement of the presternum is two and a quarter inches; that of the mesosternum almost two and three quarter inches, and of the xiphisternum one inch and a quarter. Beyond the lower bony extremity of the mesosternum is a narrow piece of cartilage, on either side of which is the articulation with the sixth costal cartilage. Continuing it downwards is the xiphisternum with an oval bony centre in its upper part. I should imagine that the specimen was obtained from a subject of between twenty-five and thirty-five years of age.

The lower costal cartilages present some unusual abnormalities, which I have also attempted to figure. They point to an undeveloped condition of the lower part of the bony and cartilaginous framework of the thorax, and I hope to show later that this is what one finds in cases in which the upper portion of the chest has been increased in size, owing to the presence of cervical ribs, or from some cause which is not so apparent. As will be noticed the left ribs retain, throughout, their lower level. The fifth costal cartilages are firmly united and continuous with the sixth, especially on the left side. This union on the right side is somewhat movable, and presents in its centre on section a synovial cavity. On the left side the synovial cavity is smaller, and the cartilages are continuous around it with one another.

The sixth presents similar articulations with the seventh costal cartilages. The latter on the right side ends in an obtuse extremity, which is continued on as a fibrous cord. This blends with the perichondrium of the sixth cartilage just below its articulation with the sternum, and on the left side the cartilage blends with the lower margin of the sixth, about two inches below its inner extremity. The seventh and eighth cartilages are united in a similar way.

At some of these junctions the cartilages are completely continuous, while at others the central opposing surfaces of cartilage are lined by a synovial membrane. This is all merely an exaggeration of conditions found normally between the lower cartilages of the ribs.

Examining this specimen, I think there is no doubt that the cartilages, which articulate with the presternum and mesosternum at their junction are the normal second costal cartilages, and that the broad cartilage attached to the presternum is formed by the fusion of the cartilages of a cervical and first thoracic rib.

The mesosternum is also abnormal in its disproportionate size, and in having articulated to it only the second, third, fourth, fifth, and a part of the sixth costal cartilages. It would be difficult to define the exact termination of the mesosternum and the upper limit of the xiphisternum, owing to the cartilaginous condition of the lower end of the mesosternum.

A point of much practical importance is the difference in the level of the cartilages and ribs on either side. This is owing in this case chiefly to the obliquity of the lower margin of the manubrium, and in the specimens of this condition I have examined, I have usually found the ribs lower on the left than on the right side. This might easily lead to error in defining the position of viscera, especially that of the apex of the heart.

Professor Turner describes a case¹ which resembles this one closely in many particulars. In it on the right side, there was a well-formed movable cervical rib, which articulated behind with the body and transverse process of the seventh cervical vertebra, and was situated immediately above the first thoracic rib, which it resembled in shape. Its anterior extremity blended with the cartilage of the first thoracic rib, and through it was connected to the manubrium. Where the rib joined the cartilage it was hard and nodulated, and its texture resembled that of ossified costal cartilage. On the left side there was a supernumerary cervical rib, which was movable and more slender than that on the right. It did not extend so far forwards, for its anterior somewhat pointed end articulated by a movable joint with a small tubercle on the upper surface of the first thoracic rib close to its inner border, 1·2 inches behind the costal cartilage. Both ribs were encrusted with cartilage at this point, and a thin interarticular disc of fibro-cartilage was interposed.

Professor Turner mentions that in this case the cervical rib,

¹ "On Supernumerary Cervical Ribs," 'Journ. Anat. and Phys.,' 1870, Case 1.

where it joined the cartilage of the first thoracic rib, presented the appearance of ossified costal cartilage. I should imagine that the rib had had a cartilage of its own, as in my case, but that that cartilage had become ossified.

In the case I have described, the process of ossification has proceeded to a great extent in the cartilage of the cervical rib on the right side, and is peculiarly limited to it, the remainder of the cartilage, namely that part which is formed by the first thoracic costal cartilage, being quite free from bone formation. The cervical rib, on section, presents an appearance identical with that of the other ribs. The ossification in the cervical costal cartilage on the left side is indicated by a circular patch of bone above the foramen. This, I believe, is merely an exemplification of the rule that the first costal cartilage undergoes osseous change long before any of the rest, and as this cervical cartilage is higher than the normal first and therefore exposed to more strain, ossific deposit has taken place in it before it has appeared in the first thoracic cartilage. On neither side does Prof. Turner make any mention of any increase in the size of the first thoracic rib or its cartilage, which must I believe have existed.

In the case of the left costal cartilage in my case, I should suppose that the cervical rib had blended with the first thoracic some way external to the cartilage. I would also suggest the possibility of these cervical ribs having articulated with an eighth cervical vertebra, and not with the seventh, as was the case in a skeleton of a silvery gibbon, which will be described later.

In the same paper, Prof. Turner describes several dried specimens in the Anatomical Museum of the University of Edinburgh, and I will tabulate them briefly here as one can then more easily perceive resemblances to those I have figured.

Specimen 883 c.—Right cervical rib movable, 1·5 inches long. Anterior free end pointed.

Left nearly three inches long, movable, and closely resembling right cervical rib in first case described, and that in my case.

First pair of thoracic ribs articulate with bodies of seventh cervical and first dorsal vertebræ.

Specimen 883 d.—Right cervical rib, movable, 3·2 inches long. Anterior end free and pointed.

Left side: No movable rib. Free extremity of posterior transverse process was thick and prominent.

Specimen 383 f.—Two short rudimentary ribs are connected to seventh cervical vertebra. They appear to be ankylosed by their tubercles to the vertebral transverse processes, but the head articulates with a very distinct tubercle-like elevation on the side of the body of the vertebra.

On the left side a slender bar of bone lies behind and parallel to the neck of the rib, and subdivides the interval between it and the transverse process into two foramina. It is continuous at its extremities with the body and transverse process of the vertebra. Each rib only projects $\cdot 3$ inch beyond vertebral transverse process. The first pair of thoracic ribs articulates only with the first dorsal vertebra.

Specimen 387.—First right thoracic rib presents a strong process, $\cdot 6$ inch in breadth and $1\cdot 4$ in height, rising from the upper surface of the shaft close to its inner border and $\cdot 8$ in front of the tubercle. It is broad and flat at its summit, and has obviously been intended for the attachment of the cervical rib.

According to Professor Wenzel Gruber, of St. Petersburg,¹ who analysed seventy-six examples of cervical ribs occurring in forty-five individuals, they usually occur on both sides of the seventh cervical vertebra, and more than one pair has not been met with in the same person. Sometimes they were ankylosed to the body and transverse process (or only to the latter) of the seventh cervical vertebra. Only seldom did they end in a distinct costal cartilage. Usually only an external intercostal muscle occurred in the supernumerary space. In Professor Turner's first case and in mine there were internal and external intercostal muscles.

Cervical ribs may be either the unusually developed rudiments of the anterior transverse process or rib of the seventh vertebra,² or merely unusually developed epiphyses which articulate only with the transverse process of the seventh vertebra. In the former case they are homologous with the inferior roots of the transverse processes in birds, and the cervical ribs

¹ 'Mém. de l'Acad. Imp. des Sciences de St. Petersburg, Tome xiii, 1869.

² This was first described by M. Hunault in the 'Mémoires de l'Académie Royale de Paris,' 1740, p. 525, 12mo, Amsterdam, 1744.

in crocodiles; in the latter with the rudimentary ribs connected with the eighth and ninth cervical vertebræ in *Bradypus tridactylus*, and with a rudimentary rib once seen by Gruber in *Canis familiaris*.

When a rudimentary rib, it may present one or other of four degrees of development:

(a) When it is very short, and possesses only a head, neck, and tubercle.¹

(b) Where it extends beyond the transverse process, possesses a body, and ends either free, or joins the first thoracic rib.

(c) Where it reaches beyond the transverse process, and is connected either by ligament or by the anterior end of its body with the first costal cartilage.

(d) Where it resembles a true rib and possesses a costal cartilage, which joins with the cartilage of the first thoracic rib and the manubrium.

As a rule the head of the cervical rib articulates with a cylindrical process or elevation on the side of the body of the seventh vertebra. Not unfrequently a process, eminence, or tuber exists on the thoracic rib at the place where it articulates with the cervical rib. Prof. Gruber considers that the rule, laid down by Halbertsma² and supported by Luschka,³ that a cervical rib 5·6 cent. and more in length supports the subclavian artery, while one shorter than 5·1 cent. does not support it is untenable, as specimens have been seen by himself and Erb in which this rule was not borne out.

I am indebted to Prof. Turner's paper on "Supernumerary Cervical Ribs" for the above résumé of Prof. Gruber's elaborate essay. The same anatomist has more recently published two cases in the seventeenth volume of the Journal of Anatomy and Physiology, illustrating the higher grades of cervical costal arch. In the first case, the movable left cervical rib, whose shaft was 1·4 inches long, terminated in a point from which a fibrous band passed forward to join a cartilage 0·6 inch long, which fused with the cartilage of the first thoracic rib immediately below the right sterno-clavicular joint. On the right side the cervical rib had a head, neck, and

¹ Of this class I have seen several examples, but all fixed.

² 'Arch. f. d. Holland Beiträge,' Utrecht, 1858.

³ 'Denk. der Kaiserl. Akad. der Wissenschaften,' Wien, 1859.

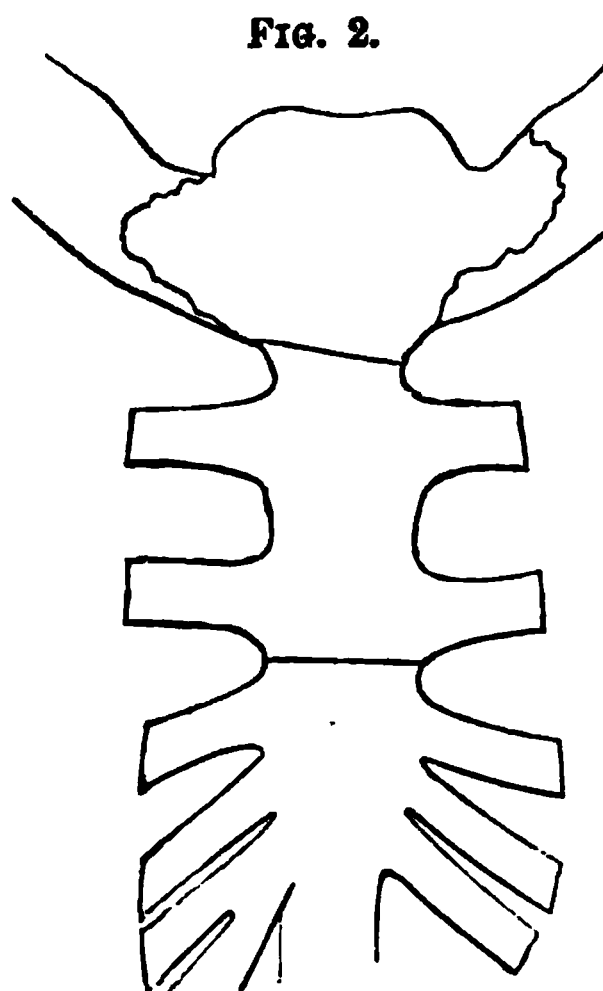
tubercle and a slender shaft 0·9 inch long. This ended in a point from which a fibrous band passed forward to be attached to the first rib behind the scalenus anticus.

In the second case the right cervical rib was movable. It had a shaft 1·4 inches long, pointed and connected by a fibrous band to the first thoracic rib, where cartilage and bone became continuous with each other. The first rib was increased in breadth at this point to 1·4 inches.

On the left side there was a movable cervical rib with a shaft 1·2 inches long. Its pointed end joined by a fibrous band a costal cartilage, which was parallel to and immediately above the cartilage of the first thoracic rib with which it blended prior to the attachment of the cartilage to the manubrium. The greatest breadth of the thoracic rib was 0·9 inch.

This case resembles the next specimen I shall now describe in the increased breadth of the first thoracic rib and in many other points.

Fig. 2 represents the sternum, first ribs, and cartilages



obtained from an adult male subject in the dissecting room of Guy's Hospital.

The first thoracic ribs are very massive and large and have the usual obliquity and direction of the first rib. The measure-

ment of the right rib along its convex margin is 8 inches, along its concave or inner margin 5 inches, while that of the left along its convexity is $7\frac{3}{4}$, along its concavity $4\frac{3}{4}$ inches. The span or a line joining its extremities measures $2\frac{1}{4}$ inches. The rib reaches its greatest breadth of $1\frac{1}{2}$ inches at its anterior extremity. The markings for the attachments of muscles are very distinct. There is no prominence corresponding to the scalene tubercle, nor is there any elevation along the margin receiving a fibrous prolongation from the cervical ribs. The subclavian artery and vein passed over its upper surface. The head of the rib is large and articulates by dense fibrous tissue with the body of the first dorsal vertebra, the lower margin of the body of the seventh cervical, and the intervening fibro-cartilage. This articulation with the seventh cervical is merely owing to the large size of the head. This, however, is not out of proportion to the rest of the rib, which is enlarged throughout.¹

The costal cartilage is $1\frac{1}{2}$ inches broad, being ossified at its outer and lower part. In the substance of each cartilage, running parallel to the oblique outer margin of the manubrium, there is an almost complete arthrodial articulation leaving a cartilaginous layer $\frac{1}{8}$ inch thick covering the manubrium. In one or two points the cartilage is not quite divided, but the movement of the sternum on the cartilage is pretty free. The joint is surrounded by a dense fibrous capsule especially on its posterior surface.² There are two cervical ribs, the right one movable and the left fixed. The right is $1\frac{1}{2}$ inches long, and articulates by its head with a prominent tubercle in the centre of the side of the body of the seventh cervical vertebra, being united to it by an intervening fibro-cartilage and by a fibrous capsule. Its tubercle articulates with the enlarged transverse

¹ I find that it is by no means rare for the head of the first thoracic rib to articulate with the lower part of the body of the seventh cervical vertebra and the subjacent fibro-cartilage.

² This form of articulation I have found to be developed (in healthy flexible first costal cartilages) at an early period of adult life in subjects in whose sterna the manubrium and gladiolus are so firmly united as to allow of very slight if any movement between these bones. This form of articulation must not be confused with that which develops at a late period in ossified first costal cartilages to serve a similar purpose. Cf. "Fracture of Sternum with Costo Chondral Disloc.," 'Trans. Path. Soc.,' 1883, and "Costal Assymetry," 'Journ. of Anat. and Phys.,' 1884.

process of the seventh cervical vertebra. The rib ends by a rounded free extremity among the scalene muscles, and has no fibrous band continuing it and attaching it to the first thoracic rib.

Connecting the side of the body of the vertebra behind the tubercle for articulation with the rib and the transverse process is a slender bar of bone, which subdivides the interval between the neck of the rib and the transverse process. A precisely similar condition is present in 383 *f* described by Prof. Turner, but in that the rib was not movable. In reference to this point, I would call attention to a case described by the same author in the 17th volume of the 'Journal of Anatomy and Physiology,' "A first dorsal vertebra, with a foramen at the root of the transverse process," as I think together these specimens explain one another somewhat.

In that paper he describes a first dorsal vertebra presenting in the root of the left transverse process a vertical foramen immediately in front of the articular process, and a deep notch in a corresponding position on the right side. This notch in the recent state was converted into a foramen by a ligamentous band.

The seventh cervical vertebra presented a foramen in each transverse process.

In the fifth, sixth, and seventh on the right side, and in the sixth on the left, the vertebrarterial foramen was divided into two parts by a slender bar of bone.

These cases would lead one to suppose that this process of bone was derived from the transverse process proper, and not from the anterior or costal process.

On the left side, the rib presents only a head and neck. The former is fixed internally to a tubercle similar in everything to that on the right side. Between it and the head of the rib is a fissure, and surrounding this is a fibrous capsule. The neck externally ends in the transverse process, which is even longer than that on the right side, being $1\frac{1}{2}$ inches long.

The vertebral formula is normal as is also the number of the thoracic ribs. The manubrium measures 3 inches, the gladiolus 4, and the xiphoid cartilage 1.34.

The seventh right costal cartilage is united by a bony deposit about $3\frac{1}{2}$ inches from its inner extremity to the cartilage of the sixth rib. It then becomes free, running parallel to the

sixth cartilage for $1\frac{3}{4}$ inches, when it forms by uniting with it a common cartilage, which is united to the lower extremity of the mesosternum.

The seventh left costal cartilage terminates in a blunt extremity about 2 inches from the sternum. For the inner 2 inches of its course it is firmly united to the sixth costal cartilage by a dense fibrous capsule, the approximated surfaces forming a synovial cavity.

The eighth, ninth, tenth, and eleventh ribs are much smaller than usual, and the twelfth ribs measure only $1\frac{3}{4}$ inches in length. Besides, they are more than normally united to one another by cartilage and ligamentous tissue, the whole tendency being to diminish the lower portion of the chest cavity. This is also well seen in the relative sizes of the manubrium and gladiolus.

The subject in its transmission from the Infirmary had sustained some violence, as a result of which the manubrium had been broken transversely just below the attachment of the large first costal cartilages, the line of fracture being from above obliquely downwards and backwards. The fracture in this subject, owing to the exaggerated size of the first cartilage, shows well the great influence that this cartilage has in causing fracture of this bone and in determining its position. The sternum was also broken transversely midway between the attachment of the third and fourth cartilages. I have seen no record of this exaggeration in the proportions of the first rib and cartilage accompanying presence of cervical ribs in man, except in one of the cases more recently described by Prof. Turner. In my case the condition is fairly symmetrical.

The lower ribs on the right side are, however, distinctly smaller than those on the left side. This is what one would expect, as the cervical rib on the right side was better developed than the left one, and consequently the right first thoracic rib is larger than the corresponding one on the left side.

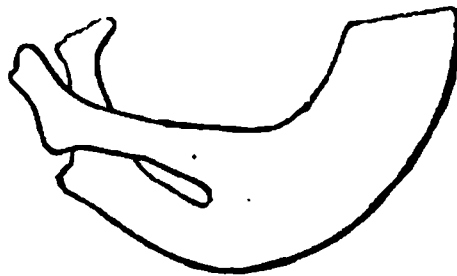
The tendency in the two cases I have described to an increase in the size of the upper ribs and cartilages, and to an exaggerated articulation and union of the lower ribs and cartilages with one another, and to a diminution of the number of ribs articulating with the sternum and to a relative shortening of the gladiolus, is well marked.

The condition in the last case resembles very much that which is present in the great armadillo (*Priodontis gigas*), where the first sternal rib is very short and incorporated with the vertebral rib, without the presence of any articulation.

Van Beneden¹ describes this increase in breadth of the first rib and cartilage as occurring in a porpoise. On the right side a cervical rib articulated in front with the sternum, while a left cervical rib did not reach the sternum, but the first thoracic rib widened out at its sternal end, and was connected to that bone by two distinct cartilages.

In the museum of Guy's Hospital I find a specimen, fig. 3,

FIG. 3.



which almost exactly resembles Case 3 in Prof. Turner's paper in the vol. xvii of 'Journal of Anatomy and Physiology.' It has no history or description beyond that it is probably a fused cervical and first thoracic rib. It is a right rib. In its anterior two inches it resembles in form and in the horizontal direction of its surfaces the normal first thoracic rib. It is uniform in breadth in this part. At its termination it is $\frac{7}{8}$ inch in diameter. Its extremity is hollowed and contains a remnant of the costal cartilage. It presents no indication of a scalene tubercle nor any prominence representing any tendency to the completion of a costal arch. Continuing the direction of this portion backwards is the lower branch of the bifurcated ribs, so much so that if the upper branch were removed, the remainder would be apparently a normal first rib.

The lower piece, before its union with the upper, presents a head, about the usual size of that of a normal first rib, a neck an inch long, and a tubercle with a facet and a shaft also an inch long. The greatest breadth of this portion of the shaft is $\frac{3}{4}$ inch, while the greatest breadth of the anterior portion of the rib is a little more than an inch.

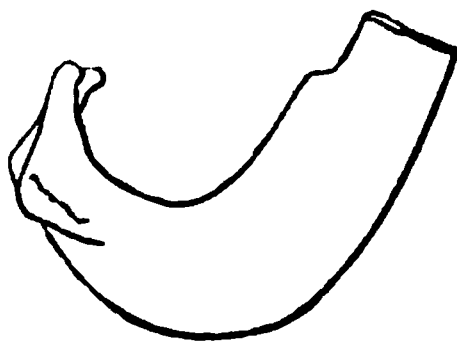
¹ 'Bulletin de l'Acad. Roy. des Sciences de Belgique,' xxvi, p. 7, 1868.

The upper limb has not the curve and direction of the lower. Its shaft is quite straight, thick, and rounded, being directed forwards, downwards, and slightly outwards to its point of union with the lower limb.

The tuberosity is large, presenting a large, hollow, transversely oval facet with internally a prominent, sharp, bony margin. The neck is less than half an inch long, and continues the direction of the shaft. It ends in a rounded, smooth extremity. The length of the rib measured along its convex margin is just $5\frac{1}{2}$ inches, the space from the head of the lower limb of bifurcation $2\frac{1}{4}$, from the upper $2\frac{3}{8}$. These measurements correspond to the average measurements of the normal first rib, rendering it probable that the specimen is one in which the right cervical rib has become fused with the first thoracic. It would come under Prof. Gruber's second class.

The next specimen, 1045, fig. 4, is also in the Guy's Hospital

FIG. 4.



Museum. It differs somewhat from the last. In this two ribs have become fused together. The heads, necks, and portions of their shafts ($\frac{3}{4}$ inch of the upper and one inch of the lower) are free, and follow exactly the same curve. The heads are but slightly larger than the necks, and present round, somewhat convex facets. The necks are one inch long and the tubercles are prominent, smooth, and convex. The shafts are $\frac{1}{2}$ inch broad. The common shaft is not horizontally placed as in the last specimen, but is oblique, so that the outer border is lower than the inner. At the back part it is $1\frac{1}{4}$ inches in breadth and reaches its maximum $\frac{3}{4}$ inch from its extremity. This is due to a sudden cessation of the upper rib. The angle formed here is smooth and rounded, and shows no indication of the attachment of a cartilage or fibrous band. Beyond this point the shaft measures $\frac{7}{8}$ inch, which was the measurement of the anterior extremity of the rib in the last case. In it there is a

depression containing a piece of the costal cartilage. Along the inner margin from the head to the point of sudden diminution in breadth of the shaft is 3 inches, and the span $1\frac{3}{4}$ inches. The span from the second head is $2\frac{1}{4}$ inches. Measurement along convexity is $8\frac{1}{4}$ inches, being less than that of the normal second rib, which varies between $8\frac{1}{2}$ and $9\frac{1}{4}$ inches.

I believe this is an instance in which the cervical rib has become fused to the first thoracic, though some arguments may be urged against it. Prof. Turner considers that fusion of a cervical and first thoracic rib is extremely rare, and he regards most of the cases described by anatomists as being examples of union of the first and second thoracic ribs. In this conclusion he says he has been much supported by Cases 1 and 2 in his last paper.¹ Case 3, which he describes, and which I have mentioned before, he thinks is an example of the rarer abnormality. At the same time I cannot help thinking that in his criticisms he errs in laying too much stress on the direction of the surfaces, the breadth of the rib, and its measurement along its convex margin. These vary so much, even in the typical normal chest, and still more when the balance is affected by an exaggerated development in the upper or lower part of the chest. One must regard the chest wall as a whole, and not attempt to generalise from the examination of only a part.

Looking at the second case which I have figured (fig. 2), in which the first ribs and cartilages are enormously enlarged, the measurement along the convexity of one rib being 8 inches, and Case B in Prof. Turner's paper,¹ where the left first thoracic rib became broadened to 1.4 inches, after the cervical rib had been connected to it by a fibrous band, and to figs. 3 and 4, and to Van Beneden's porpoise, I should suppose that the same cause which determined the presence of a cervical rib also caused an increase in the dimensions of the first thoracic rib and cartilage, while at the same time it caused a want of development of the lower ribs and costal cartilages, and as in figs. 1 and 2, a shortening of the gladiolus. It also produced a diminution in the length of the twelfth rib, as in Case A

¹ "Cervical Ribs and so-called Bicipital Ribs in Man." Vol. xvii, 'Journal of Anatomy and Physiology.' If one had the sternum as well as the fused ribs to examine, one might decide in favour of the presence of cervical ribs, if the number of cartilages articulating with the sternum was diminished, and *vice versa*. To this I will refer again later.

(Prof. Turner, 'Journal of Anat. and Phys.' vol. xvii), where these ribs were 2·8 inches long on the right side when the cervical rib had a shaft 1·4 inches long, and 1·4 on the left side, the cervical rib having a shaft ·9 inch long; and in Case B, where the shaft of the right cervical rib was 1·4 and the twelfth rib 1·5 inches, the shaft of the left cervical being 1·2 and the twelfth rib 2·25 inches; and in Case 1 ("On Supernumerary Cervical Ribs," Prof. Turner, 'Journal of Anat. and Phys.' vol. ii), where there was a large cervical rib on the right side, the cartilage of which joined that of the first thoracic and a smaller left one, the twelfth ribs were merely represented by elongated costal transverse processes. Again, in the second case I have described (fig. 2) the right cervical rib was free and about $1\frac{1}{2}$ inches long, and the left was fixed, while both twelfth ribs measured $1\frac{3}{4}$ inches and articulated by a tuberosity with the transverse process of the twelfth dorsal vertebra, the right being smaller than the left.

On the other hand, I would suggest that the same influence that determines the want of development of the upper part of the chest causes, firstly, a diminution in size of the upper ribs, with a tendency to fusion of the first and second, as shown by its span and by the measurement along its margins being diminished. Take the Cases 1 and 2 in the 'Journal of Anat. and Phys.,' vol. xvii. In Case 1 the measurement along the outer margin of the second rib was eight inches, and in Case 2 it was $8\frac{1}{2}$ inches, the normal variation being between $8\frac{1}{2}$ and $9\frac{1}{2}$ inches.

Secondly, it causes an increase in the length of the mesosternum as shown by its length and proportion to the prester-num,¹ and in the number of costal cartilages articulating with it, as may be seen in fig. 6, where the manubrium is seen to be $1\frac{3}{4}$ inches long and the gladiolus $4\frac{1}{2}$ inches long, and on the right side eight cartilages articulate with the sternum.

This condition is also seen to be more marked on that side in which the fusion is more advanced.

The fusion of the ribs is caused, I believe, by the arrangement of the centres in the sternum, especially the direction of the lower margin of the centre in the manubrium.

¹ The average length of the manubrium I find to be $1\frac{1}{2}$ to 2 inches, and of the gladiolus to be 4 inches. The xiphoid cartilage is very variable in length.

Thirdly, it causes a lengthening of the lower ribs, and is best exemplified by the length of the twelfth ribs, as is well seen in Prof. Turner's Cases 1 and 2 just referred to. In Case 1 the twelfth ribs were each about four inches long, and in Case 2 the right twelfth rib measured 3 inches and the left $3\frac{1}{4}$, and in both these cases there was fusion of the first and second thoracic ribs. The ultimate cause at work in the production of these effects is probably the necessity of a certain amount of lung to the individual, so that if the upper part of the thoracic cavity be enlarged by an increase in the size of the upper ribs, the lower part is diminished and *vice versa*.

The following specimen is also obtained from our Museum, and fig. 5 is a fairly good representation of it. In it, I believe there is fusion of the first and second thoracic ribs. It is peculiar in its great breadth and in the thinness and incurvation of its lower part.

It resembles specimens 383 *a* and 80 *c* in the Anatomical Museum of the University of Edinburgh. They are figured in vol. iv of 'Journ. of Anat. and Phys.,' in Turner's article on the "So-called Two-headed Ribs in Whales and in Man." In that paper Prof. Turner regarded them as instances of fusion of the first thoracic with a cervical rib, but in his most recent publication on the subject, he concludes that the ribs fused are the first and second thoracic. The points which made him alter his opinion were the similarities of these specimens to those in Cases 1 and 2 in the same paper, and also the close resemblance in the form of the heads and necks, in the place of fusion of the shafts just outside the tubercles, in the obliquity of the surfaces of the common body, and in the presence of a roughness for the attachment of the serratus magnus. Also in the approximation of their measurements to those of the first and second thoracic ribs.

The condition that I should regard as the most important is the mode of division of the common shaft in front, and the attachment to these divisions of costal cartilages which approximate in breadth to those of the first and second cartilages. If, for instance, in fig. 5 one looked upon the lower division as representing the first rib and being continued on by a costal cartilage to the side of the manubrium, what would have happened to the cartilage attached to the upper division,

which is separated by such an interval, viz. an inch from the lower?

FIG. 5.

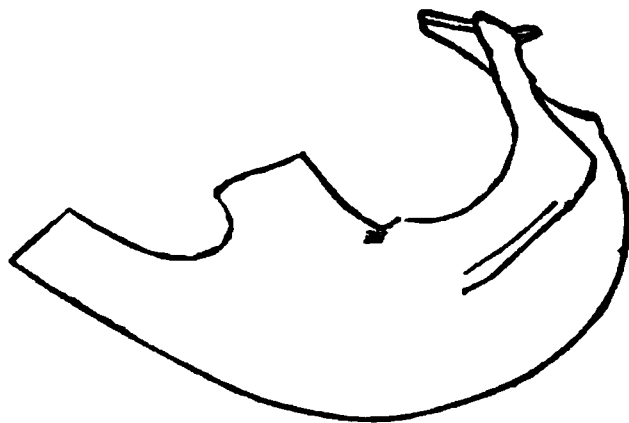


Fig. 5 then shows two left ribs blended with one another. The heads, necks, tubercles, and $\frac{1}{4}$ inch of the shafts are free. The head of the upper presents an oval flat facet $\frac{1}{2}$ inch broad, that of the lower being more than $\frac{3}{4}$ inch in breadth. Their necks are about an inch long and are almost parallel to one another, allowing for the smaller arc of the upper rib. The tubercles are prominent and smooth. The shaft formed by their union is $1\frac{1}{2}$ inches broad, increasing in breadth anteriorly, reaching its maximum of $2\frac{1}{4}$ inches at the bifurcation, which takes place $1\frac{1}{2}$ inches behind the termination of the lower rib. The upper rib extends $\frac{1}{4}$ inch beyond the bifurcation, which takes place $1\frac{1}{2}$ inches behind the termination of the lower rib, and has its concave end occupied by the remains of the cartilage. It is $\frac{3}{4}$ inch broad here. The lower rib terminates in a similar manner, and is of the same diameter.

The measurement along the convex margin of the common rib is $8\frac{1}{2}$ inches, along the convex margin of the upper rib $5\frac{1}{4}$ inches, being somewhat in excess of the corresponding measurements in 80 c and 383 a (Edinburgh specimens) and Cases 1 and 2 of Prof. Turner's paper. The measurements in these cases along the convex margins of the common ribs were respectively $7\frac{2}{3}$ inches, 8 inches, 8 inches, and $8\frac{1}{4}$ inches.

On the outer surface of fig. 5 the shafts of the ribs after fusion still remain very prominent, being separated from one another superficially by a deep groove. The upper rib presents a tubercle corresponding in position to the scalene tubercle, and grooves in front of and behind this. The inner surface of

the common shaft is concave and smooth and presents no irregularity or ridge of any sort.

I am indebted to Dr. Goodhart's kindness for permission to publish the accounts of these three museum specimens.

FIG. 6.

The next specimen (fig. 6) was sent to me from the post-mortem room, but, as in fig. 1, circumstances prevented my making an examination of the rest of the body, so that the description is unfortunately incomplete.

The drawing represents a vertical section, and one remarks that the manubrium is broader than usual, being $1\frac{1}{2}$ inches broad, that the gladiolus is much longer than normal, being $4\frac{1}{2}$ inches long. These measurements present a marked contrast to those of the manubrium and gladiolus in cases of cervical ribs.

The apex of the gladiolus is considerably to the right of the middle line, and the eighth costal cartilage articulates with it on the same side, while on the left it terminates in a pointed extremity as usual.

This is an excellent example of the rule I have already formed, and I would suggest that in this case an extra lateral bony centre has appeared in the right half of the sternum, so allowing room for the attachment of the eighth cartilage. The manubrium and gladiolus are united by dense fibro-cartilage,

there being no cavity formation in it. Most of the lower cartilages, viz. the right third, fourth, fifth, and sixth, and the left third and fourth, are united to the sternum by complete arthrodial articulations, those below them by fibrous tissue alone. Between the gladiolus and the lower half of the right second cartilage and the left united cartilages are small synovial cavities. The first and second right cartilages seem fairly normal. They are perhaps nearer to one another than usual.

Those on the left side form a cartilage $1\frac{3}{8}$ inches in breadth and attached internally to the entire margin of the manubrium, to the fibro-cartilage between the manubrium and gladiolus, and to the gladiolus by dense fibrous tissue, in the centre of which there is a small synovial cavity. That this consists of the cartilages of the first and second ribs, I think there can be no doubt, if one observes its mode of attachment to the sternum. The appearance produced by the union is similar to that produced by the union of the cartilages in fig. 1.

I do not find described anywhere a case in which the cartilage was nearly as broad as in this. In Case 1¹ in Prof. Turner's paper, on the left side the fused rib did not bifurcate, but diminished in breadth to .8 inch, ending in a costal cartilage which was joined to the sternum at the junction of the manubrium and mesosternum.

As a rule, in the case of these fused first and second thoracic ribs, they either divided or presented a tubercle at the upper border.

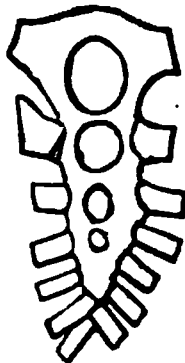
In the first instance there may be two cartilages, or the upper may be partly replaced by fibrous tissue. The lower cartilage seems to be always present and to be inserted into the manubrium and gladiolus at their junction. When there is only a tubercle there is usually a fibrous band connecting it with the upper part of the presternal margin, or sometimes there may be some portion of a cartilage. The shaft of the rib then usually diminishes in breadth in front of the tubercle, and is continued on by a cartilage which articulates with the sternum at the junction of the first and second pieces.

The following specimen (fig. 7) I have placed last as it is so incomplete, yet as it bears on what has been discussed, I will

¹ "Cervical Ribs and so-called Bicipital Ribs." Prof. Turner.

describe it now. Fig. 7 is a drawing of the cartilages and sternum of a young child seen in vertical section.

FIG. 7.

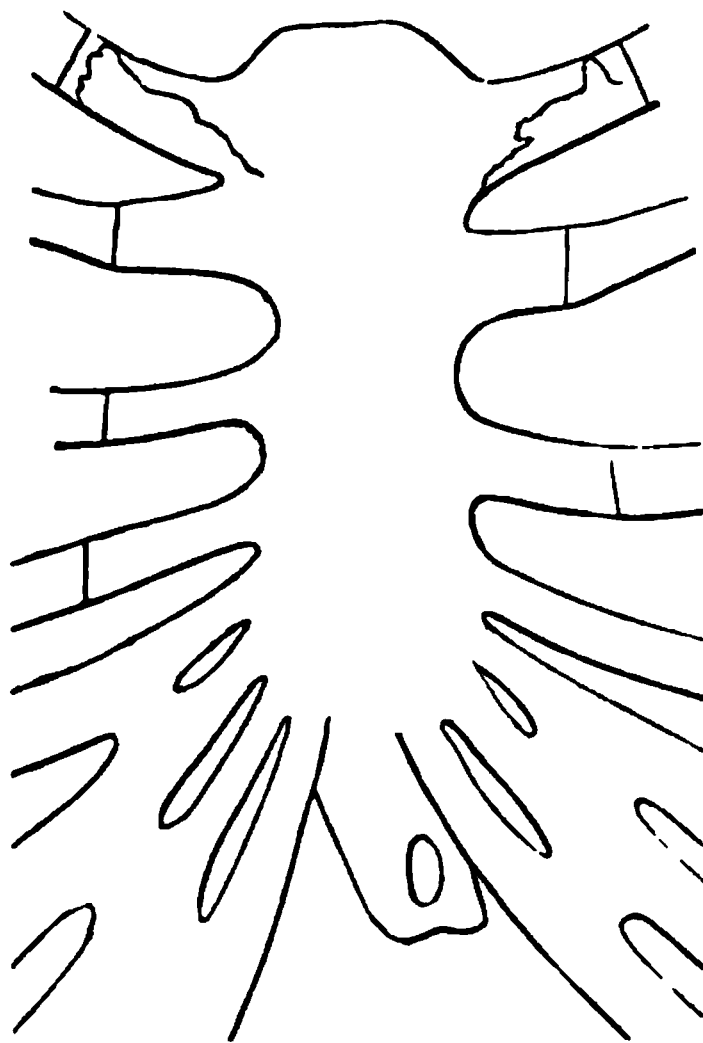


The manubrium measures $\frac{7}{8}$ inch and contains a single bony centre. The gladiolus measures $1\frac{1}{2}$ inches and contains three centres which are not arranged in quite a vertical series. The first cartilages, as far as they are present, appear quite normal. The right second cartilage, at its attachment to the sternum, is $\frac{3}{8}$ of an inch broad, but $\frac{1}{4}$ inch outside this point its breadth has increased to $\frac{1}{2}$ inch. The left second cartilage is $\frac{5}{16}$ inch broad, and is on a lower level than the right. The left third cartilage is also lower than its fellow. The fourth and fifth pair are on the same level. The left sixth cartilage articulates with the extremity of the sternum, while the right sixth articulates partly with the extremity of the gladiolus, but chiefly with the left sixth and seventh cartilages. The left seventh costal cartilage articulates with the right sixth and seventh, the right seventh with the lower part of the extremity of the left seventh costal cartilage. The xiphoid cartilage is absent, and there is evidently an absence of the lower centres in the gladiolus. The manubrium is very broad and forms an unusually large proportion of the whole bone. Applying to this specimen the preceding general rule, I should suppose that in this case the upper portion of the chest had been very large, and there appears also to have existed some change in the cartilage of the right second costal cartilage; but what was the condition of the remainder of the bony and cartilaginous framework it would be impossible to say.

Since I wrote this paper, I found in the dissecting-room at Guy's Hospital the following very interesting case of partial fusion of the first and second costal cartilages on one side, and

as it strongly supports the statements I made, I am appending it, along with two other specimens, to make the subject more complete. Fig. 8 is a drawing of the sternum cartilages.

FIG. 8.



The vertical measurement of the manubrium is two inches. That of the gladiolus is four inches. The xiphoid cartilage is long. The cartilage of the first rib on the right side is fused to that of the second at a distance of $1\frac{1}{2}$ inches from the median line of the sternum, while the distance between the margin of the manubrium on the left side and the middle line is only $\frac{3}{4}$ inch, so that the right first and second costal cartilages are fused during $\frac{3}{8}$ inch of their length. There is extensive ossification of both first cartilages, extending outwards from the manubrium.

The two first ribs are almost exactly alike in their shape and measurements. At a point corresponding to where they are crossed by the clavicle they are bent a little so as to present a concavity on their upper surface with a corresponding convexity on their lower. This gives them the appearance of having yielded at this point to downward pressure from the clavicle.

At their anterior extremities they are $\frac{3}{4}$ inch broad. Their point of greatest breadth, which corresponds to the position of the scalene tubercle, measures one inch. Their chords measure $2\frac{3}{8}$ inches. Measurements along convexity $5\frac{1}{4}$ inches. Measurements along concavity $3\frac{3}{4}$ inches. The second ribs are somewhat narrower than usual behind, and in front they are diminished to $\frac{7}{8}$ inch. Before their union with the cartilage they increase slightly in breadth. The measurement along the convexity of the right second rib is $9\frac{1}{4}$ inches; and along the left second rib $10\frac{1}{4}$ inches. The cartilage of the second rib on the right side is attached to the sternum much higher than that on the left side, being inserted on a level with the interval between the first and second costal cartilages on the left side.

The third, fourth, fifth, sixth, and seventh right cartilages are all attached to the sternum at a much higher level than those on the left side.

On the right side, the eighth costal cartilage is attached to the sternum at a point corresponding to that of the left seventh cartilage.

The measurement along the convexity of the left eleventh rib is $8\frac{1}{4}$ inches, of the right eleventh 10 inches, of the left twelfth rib $2\frac{3}{4}$ inches, and of the right twelfth rib $3\frac{1}{4}$ inches.

This bears out exactly what I have already said, namely, that when the upper part of the chest is small, fusion of the first and second ribs being present or not, the lower part of the chest is increased in size, especially on the side in which the undeveloped condition is most marked. This is shown by the great length of the lower ribs and cartilages, and by the attachment of an extra cartilage to the sternum on the right side. The difference in the relative sizes of the mesosternum and presternum is not so marked in this as in the previous cases. This is owing probably to the condition of the fusion being but partial.

The fusion is, I think, in this case determined by the obliquity of the lower margin of the manubrium, which one sees so frequently and which in this instance is very marked.

This affects the symmetry of the two sides of the chest. To show this I fastened two strings to the scalene tubercles so as to cross the ribs in a vertical direction.

The measurement from the tubercle on the right side to the

lower margin of the second rib is $2\frac{1}{4}$ inches, to that of the third rib is $3\frac{7}{8}$ inches, to that of the fourth rib is $5\frac{1}{8}$ inches, to that of the fifth rib is $6\frac{1}{2}$ inches, to that of the sixth rib is $7\frac{1}{2}$ inches; and from the scalene tubercle on the left side to the lower margin of the second rib is $2\frac{1}{4}$ inches, to that of the third rib is $4\frac{1}{8}$ inches, to that of the fourth rib is $5\frac{7}{8}$ inches, to that of the fifth rib is 7 inches, to that of the sixth rib is $8\frac{1}{2}$ inches.

In another subject in the dissecting-room I found the seventh cervical vertebra possessed two small fixed cervical ribs.

The description of the left cervical rib in Fig. 2 would apply equally well to these. The body was that of a small woman, whose chest was very narrow and undeveloped, so that the first ribs presented no very great relative increase in size.

The measurement along the convexity was $5\frac{1}{2}$ inches, along the concavity 3 inches.

Their span was $2\frac{1}{2}$ inches.

Both eleventh ribs were small, and the twelfth ribs were absent on both sides. Vertebral formula was normal.

The sternum I was unable to examine.

The two following instances of partial fusion of the cartilages of the first and second ribs are contained in the Museum of Guy's Hospital, and are of sufficient interest to be put on record.

They also display costal asymmetry.

Specimen 1038⁷⁰ is quite a peculiar one, in that the whole sternum is very much broader than usual, and it is so bent as to be very convex anteriorly, the extremity of the convexity corresponding to the attachment of the third left costal cartilage.

It is correspondingly concave on its posterior aspect. The length of the manubrium and gladiolus is 4 inches, and their average breadth is about $2\frac{1}{4}$ inches. There is no indication of the lower margin of the manubrium. This in itself is very peculiar. The first and second costal cartilages on the right side are fused for about half an inch of their extent, forming a single deep cartilage, which is attached to the sternum. The third, fourth, fifth, sixth, and seventh right costal cartilages are attached at intervals to the sternum. The first left costal cartilage is normal. The second left costal cartilage is attached

to the sternum on a level with the attachment of the third on the right side. The left third, fourth, fifth, sixth, and seventh costal cartilages are attached to the sternum on a lower level than those on the right side, so much so that they alternate with them in position. The xiphoid cartilage is still cartilaginous, and not larger than usual. The eighth cartilage is on either side joined to that of the seventh rib, and so does not reach the sternum. The fusion and consequently diminished size of the upper portion of the chest is in this case compensated by an enormous increase in the breadth of the whole of the sternum, and not by any increase in the length of the gladiolus, &c. I have not been able to find the description of a similar case.

Specimen 1048⁹⁰.—The manubrium and gladiolus measure about 6 inches in length. The xiphoid cartilage is ossified and united to the rest of the sternum.

The manubrium measures $1\frac{1}{2}$ inches, so that the gladiolus measures $4\frac{1}{2}$ inches.

The first costal cartilage on the right side has been broken off, but at its attachment to the sternum it appears to have been fused for about half an inch with the second cartilage. This condition of fusion is a little better marked on the left side.

The lower limit of attachment of the right second costal cartilage to the sternum is a little lower than that on the left side.

Eight cartilages articulate with the sternum on either side, the third, fourth, fifth, sixth, seventh, and eighth on the left side being attached to the sternum at a level considerably lower than those on the right side, though they do not quite alternate with them. This case then follows the general law.

I would refer also to a specimen in the Museum of the Royal College of Surgeons, No. 65, Order Primates. It is the skeleton of a silvery gibbon (*Hylobates leuiscus*). It has seven cervical vertebræ, and below them, thirteen vertebræ bearing ribs. There are five lumbar vertebræ.

The first ribs are but slightly curved, being much less so than the second, and also less curved than the first ribs in another skeleton of a gibbon also present in the museum. In front they are pointed, and attached by a slender carti-

laginous and fibrous band to the manubrium behind the facets for articulation with the clavicles.

On the left side the cartilage of the second rib is attached to the sternum immediately below the clavicular facet, and in the position of the normal first costal cartilage.

On the right side the corresponding cartilage is attached at a slightly lower level.

The cartilages of the third rib articulate on the same level with the lower part of the first bone of the sternum. This first piece does not appear to represent the manubrium alone, as the sternum only presents four bony plates separated by bands of cartilage, and the cartilages of the fourth ribs are attached to the cartilage intervening between the first two bones and on the same level.

The cartilages of the fifth ribs articulate with the fibro-cartilage between the second and third pieces of the sternum, also on the same level. The cartilage of the sixth rib is attached to the cartilage between the third and fourth bones, and those of the seventh and eighth ribs are attached to the sides of the fourth piece of the sternum.

The first ribs with their cartilaginous and fibrous bands resemble very closely some of the cervical ribs which have been described as occurring in man. The point of difficulty in this case is the presence of another vertebra, and the question is then whether this should be regarded as a dorsal vertebra or as an extra or eighth cervical. The ribs certainly present all the appearances of cervical ribs, and I am inclined to regard the vertebra as being a cervical one.

Possibly in some of the cases described in man an eighth cervical vertebra may have been present, and the fact may not have been observed.

REMARKS UPON THE RELATIONSHIP
BETWEEN
THE STRUCTURAL CHANGES AND THE
CLINICAL SYMPTOMS
OF
CHRONIC RENAL DISEASE ASSOCIATED
WITH DROPSY.

By JAMES F. GOODHART, M.D.

WE shall probably all admit that the descriptions in text-books of the various forms of Bright's disease often fail at the bedside and are scarcely more reliable when placed beside the facts which are demonstrable in the post-mortem room. There is a certain rough agreement between doctrine and facts which suffices to commence with, but a further experience makes the agreement only an occasional thing, and the pathology of Bright's disease is still by no means free from much obscurity. It can hardly be free at any time from difficulties, seeing that albuminuria and dropsy, either singly or in company, are of much more frequent occurrence than any recognisable structural change in the kidney. But my present purpose is not so much to grapple with such questions as these, as to point out that some of the obscurity is dependent upon tradition which represents no doubt a definite and prior stage in the acquisition of knowledge, but which should have been, and has been by in-

dividuals, thrown over when a more extended experience has been obtained.

Now the point I am making for, the fact that seems to me too little appreciated, and that needs to be taught with more emphasis, is this ; that a text-book description of acute nephritis—scanty, full-blooded urine with much albumen and dropsy—is exceptional in the wards of a hospital. The majority of cases are a colourable imitation only ; they have dropsy, much albumen in the urine, a trace or perhaps more than a trace of blood, and the urine is of low specific gravity and of large quantity. Nevertheless such cases are for the most part styled acute nephritis because the first symptom complained of is the anasarca. But these cases stay in the wards for three or four months and ultimately leave the hospital in much the same state, except for the subsidence of the dropsy, as when they entered it: that is to say, passing a urine which is of low specific gravity and contains much albumen. Watch these cases afterwards and their history is of this kind: the dropsy gradually disappears and then from time to time reappears more or less, but they do their work and call themselves well, save for an occasional headache or pain in the back, which a hospital patient thinks nothing of. The urine, however, always remains much the same as regards the albumen, and after a variable interval, perhaps five or six or nine or ten years, they come in to die, perhaps with some sudden recurrence of general anasarca and serous inflammation, or with cardiac and pulmonary troubles, but often only pallid and uræmic, without more than the least trace of dropsy ; and the kidney is found at the inspection to be mottled, yellow, and scarred or granular on the surface.

I make no doubt that this description will be allowed to be essentially correct, and if so then it would seem that one of the following propositions is true: (1) acute nephritis of middle life (for these cases mostly occur between 25 and 50) is an intractable disease and in the majority of cases becomes chronic ; or (2) what is called acute nephritis is misnamed and this disease is insidious from the commencement. The latter proposition I believe to be the more correct.

As regards the first it must certainly be admitted that acute disease of the kidney is often intractable. It is no uncommon

thing for acute nephritis to drag on for three or four months and the albumen is often even longer in taking a permanent departure; still on the whole the improvement in these cases is progressive and gives an indication of the line the disease is taking. And, on the other hand, if we take all the cases of scarlatinal nephritis, which is acute enough and nephritic enough if we judge by all the signs which serve as evidence of nephritis in other cases, we find that the majority get well without any such very prolonged illness. It will be said, perhaps, that I have no right to take an inflammation due to a cause which has a known course and tendency to subside like the scarlatina poison, and argue from it for other forms of nephritis; but this does not appear to me to be an objection in this case, for the reason just given that the inflammation in the kidney, specific or not, could hardly be more pronounced. Moreover, though this is certainly not an argument of much solid worth, it would be a strange thing, with the evident tendency of all our tissues towards repair, if the kidney should in the large majority of cases show such a tendency to irreparable destruction as it would appear to do, if the class of cases I am now concerned with began as an acute disease in hitherto healthy organs. From this side of the question, therefore, I am disposed to doubt the correctness of our present nomenclature; and if I take its other aspect and examine the structural changes of the kidney itself and the conditions under which they are found, the conclusion at which I arrive is even more positive. Acute nephritis is a disease which is frequently associated with lardaceous disease; it is frequently associated with chronic obstruction to the outflow of urine from the bladder; it cuts off many a gouty man towards the close of his prime and when we may assume that there has been some pre-existing disease of the kidney. I might even say it is allowed by many, through the large white kidney, to end in a granular form of kidney, although this I venture to doubt.

Now what do these associations mean? Take lardaceous disease: although it is a change which is widely distributed, yet in any one organ advanced changes are often only found in this part or that; in those intervening there may be little disease or none. Further, lardaceous disease in the kidney is particularly prone to set up inflammatory changes about it:

the epithelium in the tubes undergoes fatty degeneration and the proliferative changes which are known by the name of catarrh; and the stroma becomes crowded with nuclei. Thus we have, disseminated through the kidney, centres of chronic inflammation dependent upon the lardaceous change in the vessels. And here let me make a slight digression to say how rare a thing according to my experience is pure lardaceous disease of the kidney. It is far more frequently associated with chronic parenchymatous nephritis, more or less; and consequently I am always in doubt as to the precise value of low specific gravity of the urine and polyuria as indications of the existence of the lardaceous kidney, for the two symptoms are equally those of the more chronic forms of parenchymatous nephritis.

Again, what does obstruction to the outflow of urine entail? I doubt if histology has proved itself a greater benefactor in any other organ in the body than in the kidney, and in any other disease than what now goes by the name of ascending nephritis, for I cannot conceive of a subject which has a more practical or wider bearing upon renal disease than it. What harm does obstruction to the outflow of urine do? Histology shows that ultimately it works much the same mischief as does lardaceous disease. It leads first to backward pressure in the renal tubules, the backward pressure deranges the circulation, the secreting cells suffer and the perivascular tissue becomes as we call it inflamed. From this cause the vessels become thick or swollen, the connective tissue also, the nuclei of these tissues are present in excess and the blood-corpuscles wander into unwonted paths. Here we have the simplest and mildest form of the disease, but the obstruction is often associated with inflammation of the pelvis of the kidney, and then there opens out a prospect of wilder changes in the connective tissue of the organ, and an examination of such a case reveals foci of disease here and there all through the organ which it is impossible to distinguish from those which follow the lardaceous change, or those which accompany some cases of granular kidney; but the essence of the whole disease is still its partial distribution, so that large tracts of the kidney appear perfectly sound. Nor are the naked-eye appearances less instructive. In the early stages of the milder forms of disease there is but little to find

fault with: the capsule may be a little adherent, the pelvis dilated, and the texture tough. But in the more pronounced forms large tracts of the secreting structure become involved and the surface is scarred with reddish-grey, depressed, sanded scars, which are separated by bosses of tawny yellow, the former being the areas of chronic inflammation, the latter the more healthy parts with their tubes choked by fatty epithelium. These are perhaps the extremes of active inflammation, excepting the formation of abscesses with which we are not now concerned; but we shall not have sounded the depths of the pathological process nor the possibilities of which it is capable unless we remember that all chronic inflammatory conditions when once they have obtained a certain hold upon, or position in, the tissue are essentially infective. By this I do not mean to imply any potency derived from extraneous sources, but merely that inflammatory products of some standing have a life of their own; that they worry the tissues in which they are and so induce further inflammation; and that the activity of cell life in these areas is inimical to the physiological standard of action and in favour of some perverted form. Thus in organs such as these, when once foci of chronic inflammation are established there is a risk of a gradual spread of fibrosis throughout them, and it is possible to conceive of an inflammation or a process of cell growth so slow but so progressive as ultimately to bring about a granular kidney. In this way the pathologist can see the way from simple obstruction in the urinary passages to all forms of nephritis, and I am quite familiar with the occurrence of such cases as at any rate make the existence of a granular kidney from such a cause at least possible.

I purposed to give from our clinical records some cases of a mixed form of kidney, that is to say, a shrivelled, mottled, yellow kidney associated with a gouty taint, and therefore presumably the outcome of chronic disease; but on looking over the volumes of clinical records I find so many that it seems superfluous to do so. But the fact strikes me how sharply renal disease, or rather Bright's disease, in the wards falls into two groups, the one of acute disease tending towards recovery, a group consisting almost entirely of young people; the other, also called acute or subacute, as I have already said, but apparently very little bettered by treatment and a disease of adult life. Of fifty-six

cases of acute and subacute nephritis no less than forty left the hospital passing more or less albumen and most of them after a long stay; ten only left well without albumen, and seven of the ten were under sixteen years of age.

A fact so striking calls for some explanation. What is it? Can there be this great difference between the reparative power of the kidney in youth and childhood and in adult age? Is it not rather an essential difference in the nature of the disease, that the one is really acute and the other a slow insidious form which passes for acute solely because in its exacerbations it is liable to become associated with anasarca? Assuredly there is much reason for this if—and I think the fact cannot be controverted—the disease is found to follow lardaceous disease, obstruction to the outflow of urine, and the chronic processes associated with gout.

Many of these cases are called acute solely upon the ground of the supervention of dropsy, that is the symptom which is alarming to the patient and drives him to the doctor. No doubt the combination of circumstances preceding the advent of the dropsy has undergone some rearrangement, but what the precise conditions may be which determine the onset of dropsy it is rather difficult precisely to say. Acute nephritis would seem to be one of the most favorable conditions for its production; but very acute nephritis occasionally exists without any dropsy. Moreover, it of all the symptoms is the one which lessens or disappears without any amelioration of the others, and there are even cases, uncommon no doubt, but still most of us have met with them, where extensive anasarca quite disappears within a few days and gives rise to hopes of a speedy recovery, whereas it is really a prelude to the act of dissolution. In the same way we are all familiar with the fact that the dropsy will largely subside while the severity of the disease, as judged by the amount of the albumen and the lowness of the specific gravity combined, is not one whit relieved. I believe that it is best interpreted to mean an impeded tubal circulation. This happens in very acute cases as a temporary or persistent state, and in very severe chronic cases, such as the large white kidney, as a persistent condition, and again, on and off in the course of any chronic renal inflammation we find it come and go. It is a valuable sign as indicating the necessity

for procuring the organ as much rest as possible for the time, but its value is not great as an indication of the onset of primary disease unless these reservations be attended to.

These remarks very naturally lead up to the question of treatment. I see two classes of cases, those already described as under treatment and remaining for some months with very little improvement; others amongst the outpatients who have general directions given them to keep warm, and live sparsely, directions which they in all probability never attend to, who take tonic and aperient medicines, who continue their work all the while, and so go on on for a long time. And it is difficult to say from the experience that is gained by watching similar ones more closely in the wards that they could have done better. It is always a thankless task to question the value of any particular line of treatment by drugs. He who makes a statement in favour of this drug or that, no matter what the disease, is pretty sure to find followers and advocates, but he who would attempt to find out the legitimate range of the effects of drugs for any one disease, in that in so doing he almost necessarily circumscribes the powers of the enthusiast, will probably be said "not to believe in treatment." Nevertheless it is as well to know how we stand, for unless we do, there is a certainty of our ultimate effacement by routine, and there are few things worse than that for the patient. Now, as regards this particular form of kidney, the chronic tubal or parenchymatous nephritis, I venture to question whether drugs will act upon it in any way. Is there any drug which can be said to influence in any direct way the course of the inflammation? Some will say that diuretics illustrate some direct action, and that under their use these cases improve. Others will say that under treatment of one kind or another the albumen diminishes, and I do not deny it. What I say is that these cases are relieved upon the principle of making other parts do the work and not by any directly curative action in the kidney itself. As for diuretics I believe their value is greatly overestimated in active renal disease. Years ago Dr. Moxon repeatedly tested the diuretic action of various drugs in these cases and found them excessively uncertain, not to say in most cases useless. Since then I have often tried them and with similar results, and all that one can achieve is done, so to

speak, outside the kidney by means of other viscera. Thus the skin is made to act; and the bowels; and the action of the heart and arteries is sustained beyond the normal by means of digitalis and similar drugs. This is not to say that no treatment is of use; a patient is admitted with extreme anasarca, or dropsy of his serous cavities; or œdema of the lung; a dilated heart struggling with its overwork; he has bad headache or vertigo and so on. He is wet cupped or dry cupped, wet packed, or bathed, or pilocarpine, nitro-glycerine, digitalis, or some other drug is administered, quite properly, and no doubt whatever, to his benefit. These relieve symptoms, quiet the circulation, and thus draw off pressure from the kidney; and the damaged organ goes on as before, but the pre-existing mischief is none the better, rather is it the worse for each recurring stasis that comes to pass. And we do not know of these cases getting well—we know of them going on year after year, with care perhaps for eight or ten years—always passing albumen in quantity—always ailing, but perhaps never laid up, and ultimately dying, pale, wasted, drowsy, cold, and with panting respiration, twitching muscles, comatose, but not convulsed. The disease in the kidney creeps on all the time, or progresses fitfully, until at last, however reduced the body is, the kidney is too small to do the work. And if this is the history of these cases it is not so unreasonable to ask what is the good of keeping such patients in bed for perhaps three or four months together? Has not the experiment been carried out far more than sufficiently to prove that the disease, not the symptoms, is influenced scarcely at all? Take the case of a chronic inflammation of the lung, take the various forms of phthisis, we do not put these patients to bed and keep them there; on the contrary, we think it the worst place for them unless there be intercurrent disease of one sort or another; and things are not different in the kidney. Symptoms are to be treated, and are often very successfully treated, by drugs as they arise; the headache may be controlled, the irregular heart steadied, the sickness stopped, the dropsy lessened, the anæmia in some measure combated, and so on; but the disease is there and remains there. Therefore as soon as these or any other remediable symptoms are ameliorated and a sufficient time has been allowed to enable us to arrive at an opinion that shall be

reliable that the case is really not of an acute nature, the patient, after being duly warned of the fact that his kidneys are less able to work than once they were, and that to spare them as much as possible it is necessary to be strictly moderate in diet, an abstainer from strong drinks, and to avoid all chills, may be allowed to get about and do what work he considers he can do, and enjoy his life as best he may under these adverse circumstances and the conditions they impose.

Thus far the remarks I have made have been chiefly born of a clinical fact, the intractability of most cases of renal disease in middle life; but in the present state of renal pathology it will not be out of place to say a little more upon that aspect of these questions which a ten years' experience in the post-mortem room has naturally led me to dwell most upon, viz. the relationship between structural changes in the kidney and the leading clinical phenomena with which we find them associated. To do this I shall enumerate what seem to me to be the chief types of cases as seen in the wards, as follows:

1. Cases presenting but slight or indefinite ailment; the urine containing a moderate quantity of albumen and perhaps blood, but with a specific gravity of 1015 to 1018; these abnormal conditions rapidly, perhaps suddenly, disappearing with a few days' rest in bed. These cases are very common, and if they indicate any structural change it cannot be other than the very slightest, and is probably not a general condition of the kidney, but some change in a patch of the secreting structure here or there.

The morbid anatomy of such cases as these is represented in quite a number of ways—there is the kidney for instance of *diphtheria*, which may look a little congested, but nearly always looks quite healthy—at least without any previous knowledge of the nature of the disease it would be passed by without remark as normal. Knowing the nature of the case the kidney is often searchingly criticised, and no wonder that it is found to be wanting. I might even say the same of the examination of such organs histologically. They show very little indeed, perhaps only a little nuclear proliferation here and there. A similar history as regards albuminuria often attaches to a kidney of which all that the morbid anatomist can say is that it is pale, either in patches or generally, since

there is an absence of any speckling with opaque yellow points or even muddling of the anatomical structure such as one is accustomed to look for in inflammatory conditions.

Embolism, again, is another condition which not improbably causes similar clinical symptoms, and it again, just as the diphtheritic kidney, must be taken as an instance of changes which, in a complicated system of tubes such as the kidney, are only too likely to recur now and again.

I give two examples of this class of cases which have been under my notice in the last few weeks. They will serve to illustrate some of the clinical conditions and the uncertainty we must often feel concerning the nature of the changes which give rise to them.

From the report of Mr. W. H. BOWES.—James B—, æt. 34, was admitted for pains in the abdomen, scanty urine, and breathlessness. About a month before admission he had an outbreak of herpes. He appears to have been generally out of sorts since but continued to do his work until five days before his admission. Since then he has had various nervous sensations, stiffness of his joints, and latterly swelling of his legs and scanty urine. He was very pale with well-marked sub-conjunctival œdema, but no trace of œdema of the subcutaneous tissues; the præcordial dulness was rather increased; there was a rather loud pericardial rub; the urine contained half albumen, sp. gr. 1014; the temperature was 103°. There were peculiarities about the case in that although his pulse was up to 100 when admitted, it fell next day, notwithstanding the pericarditis, to 80, and day by day until it reached 40; it was always soft, and the tracing showed it to be dicrotic.

The day after admission the urine contained a quarter albumen and the next day also. There were traces for five or six days by the picric acid test; after that none.

From the report of Mr. J. HENDERSON SELICK.—John O—, æt. 23, a japanner, was admitted for pains in the loins and head and general œdema. He had had acute rheumatism on three occasions, and upon one of these he suffered also from hæmoptysis, the importance of which will appear presently. Three weeks before his admission he had pains all over him and his legs became useless. On December 27th his face began to swell and then his entire body; he also had epistaxis. For his

general ailment he came to the hospital at the advice of his doctor. He was anæmic with a little œdema of the face below the eyes and a little œdema also of the legs. The heart sounds were normal, the sphygmographic tracing showed increased tension and the arteries at the wrist felt thick and resistant. His urine had a sp. gr. of 1023 and contained one third albumen. He was placed upon milk diet and purgatives, and had dry cupping to the loins; on the fourth day only a trace of albumen remained, and he had no subsequent return of it. During his attacks he suffered from persistent frontal headache, and he also had slight hæmoptysis, bringing up a drachm or so of pure blood.

The nature of both these cases is very obscure. Both had suffered from acute rheumatism, but there is no common relation between mild rheumatism and albuminuria. And further, there were points in each case which lent no countenance to the rheumatic nature of the ailment. I may particularly notice on this head the quite undisturbed action of the heart in the first case, although there was a rather extensive pericarditis. Nevertheless I am inclined to think that either case, so far as the kidney is concerned, might have been embolic although it may very reasonably be objected that the local patch of disease in the kidney, although sufficient to explain albuminuria of a transient nature, is insufficient to explain a general anasarca, however slight.

2. Cases in which there is a similar want of definiteness about the cause or the onset, but there is more real illness, lassitude, and pallor, but no trace of dropsy. The urine is high-coloured, perhaps smoky, in fair quantity, of specific gravity about 1015, but contains a good deal of albumen, a little blood, and often lithates in quantity. As a matter of fact I have once seen this kind of case terminate rapidly in death. In two other cases the albuminuria was very persistent but gradually disappeared after many months. I cannot say from actual knowledge, but I suspect from what I have seen, that this condition in its slow subsidence may lead to a granular condition of kidney. The actual state of kidney I have found has been normal size or moderate enlargement, smooth surface, punctiform ecchymoses, a moderate amount of speckling of the cortex with yellow points, and more or less destruction of the streaky

appearance of the section of the cortex. Histologically the appearances unquestionably vary in different cases. There is often a great deal of interstitial change in these cases, periglomerular and other, but it is not always so. I have examined some of these kidneys expecting to find much disease and have been astonished that so little has presented itself. Nevertheless it is a very interesting form of disease, because in the absence of dropsy it wants the severity which is seen in other cases, yet the clinical picture is not devoid of such evidence of considerable deterioration of health as to constitute it an insidious form of severe disease, and one, therefore, that bids fair to last a long time and to pass on into some such chronic changes as the granular kidney represents. I believe, indeed, that this is one mode of production of the red granular kidney.

John H—, æt. 19, came to me as out-patient on October 28th, 1878. He had been a carpenter and till one week previously he had been quite healthy and not a drinker. He first complained of pain in his abdomen and puffiness of his face. For two or three days his water had been noticed to be deep coloured, and he had had pain at the end of the penis and frequent micturition. He was pale, his face puffy, his legs rather œdematous, and his urine contained much albumen but no blood. Under treatment the dropsy disappeared, and though the albumen remained, he considered himself so much better that he came no more. But at the end of November the abdominal pain returned, he had several rigors, and blood appeared in his urine in quantity. He was then admitted under Dr. Moxon. He seemed ill and had pain on deep pressure in the left loin, but apart from this his only malady was urinal. He had great pain or pains and scanty urine. It was 1015, acid, with a large amount of albumen and blood. I quote now from the very careful report of Mr. G. C. Steele Perkins. The urine was loaded with blood-casts and squamous and spheroidal epithelium. There is but little more to say; his urine remained rather scanty, varying from 14 to 24 oz., sp. gr. 1015; and he had obstinate vomiting. On December 12th, it is noted that he lies in a dreamy state; urine thick and orange-coloured, sp. gr. 1020, contains blood, a quantity of albumen, and only 1.1 per cent. of urea. He was obstinately sick all the time, vomited much and died apparently exhausted.

Another case which has recently come under my notice is that of a young man, æt. 32. He had had scarlatina some years before. He had been out of sorts for more than three months after an attack of fever of uncertain nature. He had never felt well since his attack and had not regained his flesh. He had never had any dropsy of any kind. I found his urine smoky, containing a large quantity of albumen, some blood, and an abundance of granular casts.

In another case there was no dropsy, but the patient looked yellow and wrinkled and ill for some time, and the albuminuria was persistent for many months, at times in quantity.

I examined the kidney most carefully in the first of these three cases and it is no exaggeration to say that it required a careful examination to detect anything wrong. The disease was very partial and consisted almost entirely, in those parts which it attacked, of extravasation of blood into the Malpighian capsules and compression of the glomeruli proportionately, and of extravasations of blood also into the urinary tubules. In the two other cases, I picture to myself, for fortunately they are still alive and likely to remain so, kidneys which would show in like manner only disease in certain spots, the one case taken care of on such general principles as I have already alluded to, and apparently not at all influenced by special treatment and special diet though getting perfectly well, the other still hovering between recovery and a granular kidney, like the following case.

Charlotte L—, æt. 18, was admitted under Dr. Wilks on June 18th, 1881. She had had scarlatina six years before, followed by swelling of the feet, but she gave no history of a very general dropsy. From this time there is a gap in her history until the Christmas preceding admission, since when she had had sickness and shortness of breath. Her legs and eyelids had been swollen for a month only. She was very ill when admitted. There was some œdema of the legs and of the abdominal wall to a less extent. The urine sp. gr. 1010, and very albuminous. She died within a day or two, and upon making an examination of the body I found the kidneys so much contracted as to weigh $3\frac{1}{2}$ oz. only. They were red in some parts, and sandy looking or minutely granular in appearance; bossy,

white, and fatty looking in others ; on the whole fawn-coloured. One was extremely wasted and not more than $\frac{1}{2}$ to $\frac{3}{4}$ oz. in weight. The heart weighed $10\frac{1}{2}$ oz., the left ventricle being remarkably tough, although not thick.

Now looking at the extreme contraction of these organs, one could not but suppose that the shrinking had been going on all the six years since the scarlatina, and that six months before when she became worse, some fresh inflammation of the kidney supervened and produced the fawn-coloured swelling of parts which was found at the autopsy.

3. In another and no doubt common class of cases, there is a definite history of scarlatina or chill. The urine is scanty, bloody, and contains much albumen, and there is acute and extensive anasarca. The pallor is usually moderate. Such cases chiefly occur in young persons under twenty. These symptoms are associated with somewhat variable morbid appearances. There is the large, red, smooth kidney, full of blood, corresponding to the classical descriptions of acute nephritis. I should call this a rather exceptional thing. The more usual state appears to me to be a speckled or mottled appearance associated with swelling, but both in the size of the kidney (swelling) and in the amount of pallor, mottling, ecchymosis, and general disarrangement of the anatomical structure, there is much variety, and it is impossible to predict with certainty what will be the appearance of the kidney when this group of clinical symptoms is present. As regards the minute changes there is the same want of routine ; in one case the disease will be tubal, a change which shows very little under the microscope ; in another it will be peri-glomerular, that is, outside the glomerular capsule ; in another certainly intra-capsular. My own experience would lead me to say that in such cases it is more common to find an infiltrating nuclear growth situate around some of the capsules, more or fewer, and thence spreading into the adjacent parts.

4. Cases with much dropsy, much albumen in the urine, which is pale, in fair quantity, sometimes above the average, sometimes scanty, always of low density. The pallor of the patient is usually extreme. These symptoms usually correspond with what is known in the dead-house as the large white kidney,

and no doubt this group of symptoms accompanies this form of kidney fairly closely. The kidney in such a case should be large and white or more strictly a pale fawn colour, some of its surface capillaries stellate; the surface of the section will show a marked excess of the cortical layer and much congestion of the pyramids.

Histologically the changes are, as they have often been described, chiefly tubal, the tubes are choked with fatty epithelium, and thus it happens that a kidney which looks excessively bad to the naked eye shows comparatively little when hardened, mounted, and cleared by the ordinary reagents which remove fat. Here and there patches of periglomerular inflammation will be found, for I do not believe it possible for an organ to be affected so generally as in this disease and yet that the changes shall be confined to the tubes only. It is in a measure both a tubal and interstitial form of inflammation, and in the same way the glomeruli will not altogether escape, some will be turgid with blood, some with contents extravasated into the capsule, but on the whole the disease is a tubal inflammation.

5. In this variety there is no dropsy or the very slightest. There is polyuria, the urine pale, of low density but containing much albumen, and there is much pallor; death often occurs by uræmia, which may be ushered in and accompanied by a peculiar panting or heaving asthmatic respiration which is very characteristic.

With these symptoms the inspection will almost certainly reveal not a *large* but a *contracted* white kidney. The kidney may be small and fairly smooth upon the surface, but more often it is scarred over with irregularities due to contracted red parts and swollen white or fawn-coloured parts; occasionally we find a mottled smooth surface such as is found in the *large* white kidney, while the internal parts are wasted and dilated into a saccular cyst from the effects of old standing obstruction to the outflow of urine, followed by a chronic parenchymatous nephritis by way of the so-called ascending nephritis. But whatever the external form may be, the microscopical changes vary but little; roughly speaking there is an excessive degree of interstitial inflammation. The tubal catarrh and epithelial choking are never prominent features, although by no means

wanting. The changes found in this kidney, which I describe from a typical specimen before me, are as follows: a general *mélée* of inflammatory nuclei in the arterial areæ, so that, while alternate striæ of tubes and vessels are still met with, as in the healthy organ, the inflammatory growth largely encroaches upon the tubal area on either side, and runs round it beneath the capsule of the kidney to reach other striæ of disease beyond; there is undoubted thickening of the fibrous stroma of the organ; equally undoubted hypertrophy of the vessels—of their internal, muscular and outer coats. The renal tubules appear to me to undergo changes which are peculiar: some of them are destroyed; many of them are dilated, and their epithelium instead of being large and the nucleus well distinguished from the surrounding matrix, closely studded round with a large excess of round nuclei set so closely to each other as almost to touch, and in many places give the appearance of cubical epithelium such as that in parts of the looped tubes. The tubes are often filled with casts of similar nuclei, which are like enough to those found spreading into the tissue from the glomerular capsules. There are many fibrous-looking areæ which have the appearance of obliterated and thickened tubes, and it would seem that in advanced conditions such as this, a process of organisation may go on in the tubes as well as outside them, like that which has been described as an occasional result of inflammatory exudation into the pulmonary vesicles in cases of acute pneumonia.

The glomeruli and their capsules undergo equally marked changes. The capsule is one of the most marked foci of interstitial inflammation; from its outer aspect a crowd of nuclei extends into the surrounding texture, it becomes much thickened, and the tuft in it becomes a confused mass of nuclei. Some of the tufts lose their structure altogether and fill the capsule with a hyaline densely fibrillated mass, and others, but not so many, shrivel. As regards their obscuration by nuclei I must confess, notwithstanding all that has been written on the subject, that I have been unable to determine whether the condition is due to turgidity from blood, to changes in the vascular loops, or to changes in the endothelial covering of the tuft.

6. Cases with polyuria, urine of low density, little or no

albumen, shrivelled aspect, no dropsy, heart and arteries hypertrophied. These are the symptoms of the well-known "granular" or gouty kidney and with it I am hardly concerned. Still, as this paper is a record of personal experience and not a review of other people's opinions, I will say that in my opinion there are two forms of "granular" kidney, the one a pure fibrosis, the other an active inflammatory disease; and in the latter the kidney may be either atrophied or considerably larger than the average. The two forms of disease precisely correspond with hypertrophic cirrhosis and common cirrhosis of the liver, even to this extent, that neither in common cirrhosis nor in the fibrous form of granular kidney is it usual to find an entire absence of all evidences of cellular activity. Common cirrhosis almost always shows some of the cellular changes of the hypertrophic form, and the atrophic kidney in many cases shows places of more acute inflammatory change. The large red contracted kidney invariably does so.¹

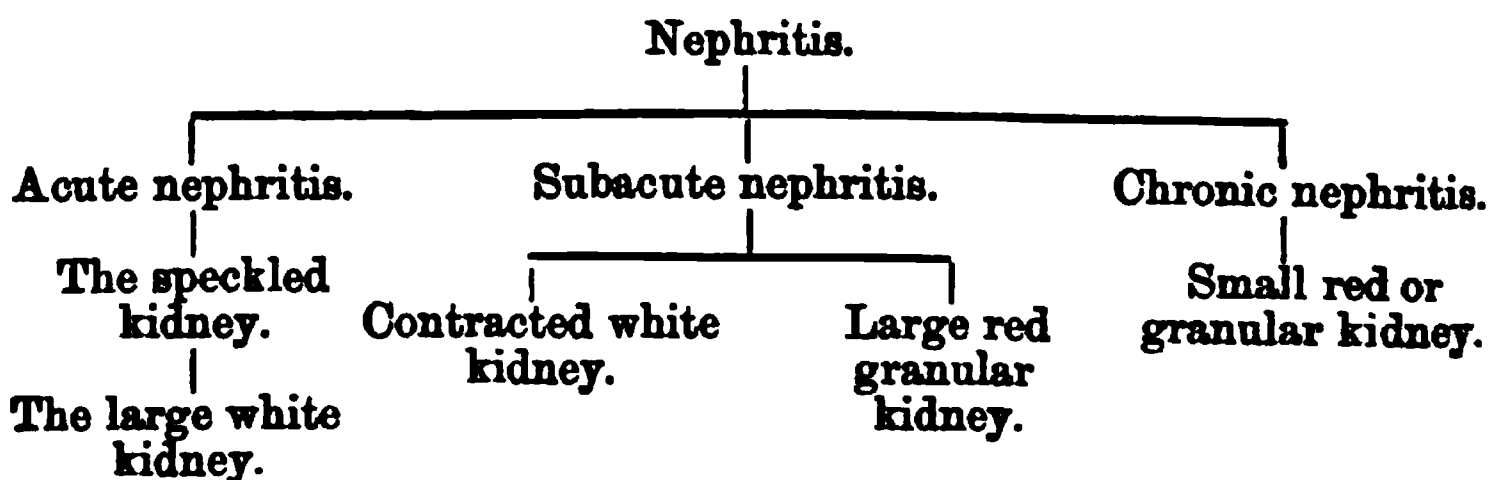
And now having passed in review the groups of cases met with in the wards and having described the kind of kidney, which on the whole is probably present in each set of circumstances, let me summarise the rough morbid anatomy of nephritis by enumerating the various forms of disease of the kidney met with in the post-mortem room.

1. A streaky or coarse kidney
 2. A pale kidney.
 3. A swollen, congested, perhaps ecchymosed kidney.
 4. The speckled kidney.
 5. The large white kidney.
 6. The contracted white kidney.
 7. The large red granular kidney.
 8. The small red granular kidney.
- } With which there may or may
} not be any clinical symptoms.

They are here arranged in no order, and it is not to be considered that any one form necessarily passes on into any other. I doubt much if there is any necessary connection between any two of them. The proper view of nephritis is to consider it all one disease, modified in the individual according to the particular arrangement taken in that case. The

¹ There is this difference, however, between the liver and kidney, that while the granular kidney is very commonly smaller than natural the liver is very seldom indeed so under any cirrhotic conditions.

elementary parts and corresponding changes are numerous, and as these re-arrange themselves in the individual so must the particular features of that case be explained. Still I should wish to say a word upon the oft discussed question whether the large white kidney ever passes on into the granular kidney. It is a difficult question because unquestionably the gouty kidney often undergoes more acute changes and then becomes mottled and fawn-coloured. Albumen also appears in the urine in quantity and the case assumes the clinical features of chronic parenchymatous nephritis; if the dropsy is considerable, as it may be under these circumstances, the case is like that of the large white kidney, and when the post-mortem room is reached there is the small white kidney. There is no history of the earlier granular changes or of ill-health, and the sequence of events seems clear, a large white kidney is there seen in course of contraction. I do not believe that this is the correct view; it is a view which is the natural outcome of the assumption that these various conditions are all stages of one disease. But I maintain that this is not the case. The varied diseases are re-arrangements of elementary lesions, and I am convinced from a careful study of the clinical phenomena and the post-mortem changes that the large white kidney comes to a stop as a large white kidney in most cases: it is the morbid expression of an acute or if not of an acute of a very generalised disease of the kidney and is a fatal affection. The small white kidney is not a large white kidney on its way towards the granular kidney but is, as I have said earlier, an insidious disease with exacerbations creeping on for years. If I might sketch out a scheme I should be inclined to put it thus:



There is yet another question which may be introduced here, viz. the existence or not of a special form, *glomerular nephritis*

After a careful examination of many specimens with reference to this point I am obliged to confess that I can see no specialty about the disease. Nor can I see for certain many of the structural changes in the glomerulus which have been stated to exist. As to the existence of the disease as a special form it so happens that intra-capsular abnormalities (and these are the most characteristic) have been sometimes most pronounced in non-scarlatinal cases. And then as to those changes themselves: most common of all is a periglomerular proliferation, which is confined to no one class of cases. There is also a confused aspect of the tuft from excessive nucleation, concerning the origin of which I am very uncertain; and there is extravasation of blood and hyaline fibrinous material into the cavity of the capsule and consequent squeezing back of the tuft to some other part of the wall, usually near the neck; in very old cases the tuft shrivels or undergoes a hyaline fibroid degeneration. I have never been able to satisfy myself of the growth of the endothelium lining the capsule enclosing the tuft. All the changes within the capsule are expressions of activity of inflammation or of sudden stasis. In the very acute cases, and therefore oftentimes in scarlatina, some fatal cases will show intracapsular extravasations, but, as I have described, others will show the turgid capillaries of the tuft and excessive nucleation; while the more chronic changes of the contracting forms will show the periglomerular changes in their most extreme degree, although it is by no means uncommon to see in the less rapidly fatal but still acute cases small islets or foci of abnormal nuclei crowding around a capsule for their centre here and there as one examines several sections of such kidneys.

This description will no doubt be said by the reader to help very little towards a classification of cases of nephritis. I admit it. It has been truly said that there are no different forms of nephritis, the disease is all one, a tune played slow or quick, loud or soft, monotonously or with varied cadence, but what I do contend for is that a careful study of the modulations of this disease as seen in the post-mortem room are of the greatest value, and help much towards the interpretation of symptoms. To take some common instances: an acute case is associated with suppression; what does morbid anatomy indicate as the

most probable changes in the kidney? I should say that there will almost certainly be found glomerular changes of some sort. If there be intermittent albuminuria long persistent after the attack, the urine being at times healthy in all respects, there is a probability of the existence of some localised damage not yet repaired, but subject to recurrent hyperæmia under press of work. If much dropsy exists, an accession of those fawn-coloured or fatty changes generally known as "tubal inflammation" is indicated; while if the dropsy subside and much albuminuria persist, that worst of all forms of kidney, the contracted white or mottled kidney, is most likely to be present.

I am quite aware of the difficulties that surround the question of the cause of albuminuria and of dropsy, difficulties sufficient to excite the interest and ingenuity of physiologist, chemist, and pathologist. But I am content here to leave to the inquirers in those other lines of investigation those elements of doubt and uncertainty which more particularly concern them and confine myself entirely to the aspect of morbid anatomy. It is this aspect which leads me to say that however dropsy may be immediately produced in these cases, it is most often associated with an increase of pre-existing tubal changes, and that its subsidence by no means indicates necessarily a return to health. There are many cases in which it represents rather some sudden check in the course of the disease, and on this righting itself or being compensated, the disease goes on as before.

The albuminuria of nephritis means severity of disease either partial or general, and there is some reason to insist upon this now that so much is rightly made of different forms of albuminuria and the early phases of disease.

I cannot conclude without saying that Dr. Wilks was the first to point out that there was no evidence that the large white kidney passes on into the granular kidney, and this thirty years ago in these 'Reports,' series 2, vol. viii, pt. 2, 1853, p. 232, "Cases of Bright's Disease, with Remarks." In that article Dr. Wilks anticipated the advance which has since been made with respect to the insidious onset of chronic parenchymatous nephritis, for I find him saying that after

anxiously seeking for patients whose cases had been reported by Bright, Barlow, and others, to see the issue, he had found that the acute cases for the most part made a permanent recovery, and those that remained ill had been chronic cases from the commencement. Perhaps I may also say that Bartels, in commenting upon the exhaustive nature of this paper, points out how curiously Dr. Wilks's work in this direction has been hitherto overlooked, at any rate by German writers.

NOTES
OF
GYNÆCOLOGICAL OUT-PATIENT CASES.

BY P. HORROCKS, M.D.

THE following cases have been selected out of a large number under treatment at Guy's Hospital during the last nine months of the year 1883.

The plan adopted in the gynæcological out-patient department has been the following :

The history of the patient has been taken and written down on the out-patient letter by the clerks or by the resident obstetric assistants, special attention being paid to the menstrual history, the history of each, if any, pregnancy, and of each lactation ; vaginal discharges ; troubles of micturition and of defæcation ; pains, more particularly bearing-down pains.

The family history has also been inquired into and any previous illness which the patient may have had. The date of the last menstrual period and of the last confinement has been noted prominently.

On my arrival the notes of each case have been read aloud, and additions made when necessary. The patient in most instances has then been examined, and the results of the examination as well as the diagnosis written down by myself.

In case no vaginal examination is made this is stated and the reason for its omission.

At subsequent visits any alterations in the physical conditions, symptoms, or complaints of the patient are noted.

Numerous sketches are made illustrating the displacements of the uterus, tumours, effusions, &c. Moreover, in all cases of erosion of the cervix examined with the speculum, a rough sketch indicating its extent has been made, and its characters detailed before any treatment has been adopted. In this way one has been able to watch the progress and compare the results.

CASE 1. *Sponge in the vagina simulating cancer.*—M. G—, æt. 72, a cachectic-looking woman, presented herself at the out-patients, on June 21st, and complained of a very offensive vaginal discharge frequently mixed with blood, which had been going on for six months, and which was getting worse. She had lost flesh considerably during this period.

Her previous health had been good. She had had two children and one miscarriage. Since her last confinement, thirty-nine years ago, she had suffered from prolapse of the womb to a slight extent. She had been to a medical man who told her that she had a cancer of the womb, and recommended her to come up to the hospital. During the six months she had suffered pain, chiefly in the back at the lower part, and this was not relieved by sitting or lying down.

The above history, together with the patient's appearance, suggested malignant disease so strongly, that on making a vaginal examination one was not surprised to find a ragged, easily breaking-down mass, in the position of the cervix, which for the moment was taken to be cancer, and was therefore manipulated very gently for fear of producing hæmorrhage. In trying to discover whether any part of the intra-vaginal portion of the cervix was free from growth, and whether the uterus was fixed or not, the mass or a large part of it seemed to break off. This was pulled out and found to consist of a black, ragged piece of sponge, the stench from which was exceedingly offensive, and quite as bad as, though of a different kind from, that frequently met with in real malignant disease.

The cervix was now felt readily by the finger and was found to be quite healthy, but small from senile atrophy, and the uterus was freely moveable. A few loose bits of sponge which had broken off were removed from the vagina, from which flowed

a little sanguineous and highly offensive thin fluid. The vagina was thoroughly syringed out with carbolic lotion (1 in 40), but a speculum was not passed, as she was very tender.

She informed us that she had been accustomed to use a sponge to keep up the womb, placing it in and removing it herself. The last time she introduced one was more than six months ago, how much more she is unable to say, but is sure it is not seven months. She was under the impression she had removed it, and was very much surprised but delighted when told it was a sponge and not a cancer. Ordered carbolic lotion (1 in 80), and instructed how to syringe the vagina.

The following week (June 28th) she came again looking much better and quite radiant. She had passed a little blood a few times, but the discharge from the vagina had nearly stopped, and was not at all offensive. Her appetite had returned, and she felt quite well. Lotion repeated and a tonic ordered, *Mist. Cinchoninæ* ℥j, t. d.

July 12th.—Hardly any discharge and that not offensive. Feels quite well. Is gaining flesh. Mucous membrane of vagina smooth; cervix and uterus as before. No sanguis on finger. No pain on examination. Lotion omitted. Medicine repeated.

At my request she came up again in three months and reported herself. She had remained perfectly well, and had regained her lost flesh. The vagina and uterus were quite normal. She had not used any more sponges, and said she would not begin again if she could avoid it. The prolapse had never been great, and she is able to get about without any support.

CASE 2. *Hysterical pregnancy.*—A publican's wife in great indignation brought up her servant girl, æt. 21, suspecting her to be pregnant. The girl stated that she had commenced menstruation at the age of fifteen, and was regular until twelve months ago when she had amenorrhœa for three months accompanied by swelling of the abdomen, which disappeared when the menses came on.

She was regular again until four months ago, since which she has not been "unwell," though she had a slight show five weeks ago. Has noticed her stomach getting larger the last four or five weeks. It became so obviously enlarged that her mistress noticed it a few days ago, and at once taxed her with being

pregnant. She has had some morning sickness and some pains in her breasts. No difficulty with micturition nor with defæcation.

On examining the breasts they were large and flabby, but the areolæ were pink, and no fluid could be squeezed out.

The abdomen was distended at the lower part by a tumour centrally placed, dull on percussion anteriorly, resonant at the sides. It was exactly in the position, and corresponded fairly well with the size and shape, of a uterus four months pregnant, but one point about it made one suspect its real nature, and that was the sensation experienced on palpation. It was not doughy to the feel like a pregnant uterus, but more elastic and fluctuating.

On vaginal examination the hymen was intact, but the vaginal orifice large enough to permit a digital examination without any pain. The cervix was hard and normal in length and position, and not at all like that of a pregnant uterus. The fundus could not be felt bimanually because of the tumour, but the cervix could be moved from side to side quite easily. The tumour bimanually felt gave the sensation of a tense cyst. A catheter was passed ten inches into the bladder, and twenty-eight ounces of straw-coloured urine were drawn off.

The tumour had now disappeared, and the uterus was caught bimanually and felt to be of its normal size. There was no pain and no difficulty on passing the catheter, and the girl still maintained that she had never experienced any difficulty whatever in micturition, and had passed the usual quantity every day. She was ordered some Aq. Menth. Pip. ℥j, t. d.

A fortnight later she was brought again exactly in the same condition, and twenty-six ounces of urine were drawn off. She menstruated a week ago.

There are few conditions which hysteria will not simulate, so that Sir James Paget was happy in the suggestion of the term *neuromimesis*. The simulation is generally purposeless in hysteria, but it is not so in malingering. Hysteria and malingering are two different things, though frequently confounded. Hysteria is a disease, malingering an imposture.

In this case the girl simulated pregnancy, but not wilfully. She was hysterical, and the hysteria took on this particular form, which very nearly cost her her situation.

The mistress was told to see that the girl passed a proper quantity of urine, and to bring her again if the abdomen got large. She has not been since.

CASE 3. Tumour on left side of abdomen and dyspareunia, due to fæces in the sigmoid flexure.—S. H—, æt. 28, married four years, having had two children, the last eighteen months ago, came on November 17th complaining of a tumour in the abdomen on the left side, which she had noticed for the last twelve months. She stated voluntarily, and before any examination was made, that the tumour diminished in size and sometimes disappeared, every four to seven days. Lately she has had much dyspareunia. Has had a bearing-down of the womb ever since the birth of her first child three years ago. Yellow vaginal discharge for two years. Bowels always confined, no pain on defæcation. Micturition frequent but not painful.

On examining the abdomen an indefinable swelling was felt in the left iliac fossa the character of which could not be well made out owing to the rigid contraction of the muscles when an attempt was made to press down upon it. On making a vaginal examination nothing beyond laceration of the cervix on the right side and a slight prolapse when she bore down was made out. She was ordered Emp. Bellad. 6 in. by 6 in. to put over the seat of pain, and Mist. Cascarillæ Co. ʒj, t. d.

December 1st.—Felt better though the swelling was just the same. Rep. ambo.

15th.—No better. Swelling is increasing in size. An examination was now made per rectum and most of it found empty, but at the upper part projecting from the sigmoid flexure was a large hard mass of fæces. Feeling quite sure that the tumour above was due to an accumulation of fæces in the sigmoid flexure, the patient was ordered 10 grains of carbonate of magnesia and a drachm of the sulphate three times a day.

22nd.—Patient's bowels have been thoroughly evacuated, and she says a large quantity came away. She is much better. The swelling has disappeared and the dyspareunia is less. Mixture to be repeated with only half the quantity of sulphate. The patient has been several times since then but only because of the bearing down. She still takes the medicine occasionally and

bowels are now more free. The dyspareunia has entirely gone as well as the tumor.

Feb. 4th 1884.—The patient came again to day after an absence of several weeks owing to domestic affairs. Not having had her medicine the bowels have again become very costive, the pain in the side has returned, and there is again dyspareunia. The sigmoid flexure is full of feces hanging over into the upper part of the rectum. Medicine repeated.

Case 4. Dyspareunia from elongated cervix and patulous os.—M. M., aged 34, married, has five children, complains of frequent and severe bearing-down pains and a desire to micturate but only scant comes with little water. Great dyspareunia. Also pain on defecation. Menstruation as a rule every four weeks lasting five days, three diapers daily, bearing-down pain before period. After her confinements menstruation has generally commenced again within a month and has continued regularly throughout lactation. She was confined with her last child nine months ago and is still suckling, but has had regular periods six eight months.

On examination finger passed straight into the os externum, which was only about two inches from the vulval outlet. The cervical canal was patent up to the internal os and admitted the forefinger quite readily for more than an inch. There was slight laceration on the left side but not nearly enough to account for the width of the os. Moreover the os stood open as a circular aperture, and lying as it did in the axis of the vagina low down, the dyspareunia and the patency can be explained by a very obvious mechanism. The intravaginal portion of the cervix felt elongated and the fundus lay back to some extent. The anterior vaginal wall was dragged down with the prolapsed uterus. She was fitted with a Hodge's pessary in such a way that the uterus was lifted up and anteverted to some extent, the cervix being directed backwards with the os looking towards the posterior vaginal wall. She was ordered some ergot to diminish the size of the uterus if possible.

A fortnight later she came up again and said that the instrument protruded sometimes and she was obliged to push it back. On examination it was found to be in good position, but there was much tenderness about the cervix and a slightly red discharge.

The pessary was removed and she was told to go without for one week and to abstain from coitus. The following week a larger pessary was put in which turned out to be too big, but finally a suitable one was obtained and the dyspareunia has been very much less ever since. She has attended every fortnight and is still under observation, her trouble now being a sense of weight in the pelvis. So far as the dyspareunia is concerned she is well. The dilated cervical canal has contracted so that although there is still a little patency of the os externum owing probably to the slight laceration, yet the finger is arrested just within the os.

CASE 5. *Stenosis of os internum; anteflexion; sterility.*—A. C—, æt. 33, has been married for twelve years but has never been pregnant. Menstruation commenced at sixteen, always regular every four weeks, duration two or three days. Slight pain for two days before, which is relieved by the flow. The menses became very scanty nine years ago and she was admitted into the hospital, where the cervix was divided backwards. Periods afterwards lasted the usual time and she had much less pain. She complains now of a “swelling of abdomen varying in size,” besides occasional nausea and discomfort after food.

On examination nothing abnormal was discovered in the abdomen or pelvis but there was a notch in the posterior lip of the cervix, evidently the remains of the operation. The sound passed the internal os with difficulty but could be pushed on for three inches from the internal os with the concavity forwards.

The above case is interesting in several respects. First, the fact that although the cervix was slit nine years ago she has remained sterile. Secondly, that the os internum is still small, requiring force to push the sound through, and that the cervix has healed up again so as to leave a mere notch to indicate that an operation has been performed. Thirdly, that though the operation failed to alter the sterility it relieved the dysmenorrhœa for a long time. Statistics seem to prove that the operation on the cervix does not remove the sterility in the majority of cases, and that in very many the relief of dysmenorrhœa is very ephemeral.

Possibly the sterility may be explained in some of the cases

of failure by the fact that the os internum is not sufficiently incised, or, if so at the time, that it contracts so as to be as bad as ever. I have recently operated again on this patient, slitting the cervix backwards and incising the internal os. She has had one period since and it was painless. During the year no less than forty married women have come with dysmenorrhœa and sterility, and in every case without exception the os externum has been found to be small and the cervix more or less conical. Only a few have come solely on account of the sterility, but many came solely on account of the dysmenorrhœa. I operated, slitting the cervix backwards, in one case because the pain was so intolerable, and this patient has had several painless periods since; but it is not long enough ago to say whether the relief will be permanent. Certainly so far the patient has expressed her great gratitude for the benefit she has experienced, and it is interesting to note that for years she has had dilators used to the cervix at various times with only partial and temporary relief.

CASE 6. *Pregnancy complicated with a fibroid; hæmorrhages; miscarriage.*—E. B—, æt. 35, has been married seventeen years and has had eight children and six miscarriages.

Menstruation commenced at the age of fourteen, and was always regular every four weeks without pain, a moderate quantity being lost at each period. She had a miscarriage nine months ago. She complains of a constant bleeding when pregnant. She says that she has been under Dr. Galabin's care three times when pregnant for the same thing. Considers herself three months pregnant now, and she has been losing blood, in gushes, continuously for a fortnight; she has not quickened nor has she had morning sickness.

On vaginal examination the uterus can be felt bimanually, and it is enlarged, reaching a little above the brim of the pelvis, on the left side it emerges into a roundish firm mass which is in all probability a fibroid tumour. The question arose whether the uterine enlargement was due to the fibroid or to a superadded pregnancy. She said in answer to minuter inquiry that she had missed her menstrual period once; and then a month afterwards she had a bleeding which she did not think was menstrual.

Passing the sound was not considered justifiable. The os was patent, but the cervix not softened. The breasts were not enlarged, and the numerous pregnancies made the pigmented areolæ worthless as far as the present diagnosis was concerned. Considering the past history of similar hæmorrhages during pregnancy, it was decided to treat her as if pregnant. No sound was used; the patient declined to come into the hospital, so she was told to keep as quiet as possible at home and a little bromide of potassium was given. The following week she reported herself and said she was much better. In another week the uterus was decidedly larger, so that the diagnosis of pregnancy was confirmed. During the third week she had a little hæmorrhage and she did not come again for six weeks, when she told us that a few days previously she woke up and was lying in a pool of blood. She still refused to come in and the medicine was repeated. The uterus was decidedly growing and she had no doubt about her pregnancy. A month later she miscarried; she thinks she was five months pregnant. Ergot was now administered and she improved rapidly. The mass on the left side does not appear to have been materially influenced in size by the pregnancy and subsequent involution of the uterus.

CASE 7. *Discharge of air from the vagina.*—E. B—, æt. 22, married two years ago, has had one child ten months ago, which she is still suckling. Menses commenced at sixteen but have never been regular and always scanty. Has menstruated twice during lactation. Instruments were used during the labour. She came complaining of “breaking wind through the vagina at the same time as through the anus, but not at any other time.” It annoys her and is very unpleasant, because she cannot prevent its making a noise. Has also incontinence of fæces when the bowels are loose.

On examination the perinæum was found ruptured to some extent, and just within the vagina on its posterior wall was a puckered cicatrix, the rectal wall behind which was perfectly smooth. This cicatrix was due probably to the healing of the ruptured perinæum, and indeed there seemed to be linear cicatrices near the puckered part as if stitches had been used. The uterus is anteverted and slightly enlarged. Ordered Pil.

Col. co. gr. x, statim, Conf. Sennæ ʒj, omni nocte, Mist. Ergotæ ʒj t. d.

She attended irregularly for two months without much benefit, and then it was found that she was pregnant between three and four months ; the ergot was stopped and ten grains of bromide of potassium were ordered instead. She attended another three months, and latterly had a severe cold. She still has her annoying complaint but it is not so frequent.

In another case, where the rupture was more extensive, reaching to the margin of the anus, the sphincter being lacerated, the patient made the same complaint about air escaping from the vagina ; but in this case I believe the air really came from the rectum, and the two cavities, rectal and vaginal, were separated by such a thin septum that so far as the patient's sensations were concerned they formed a cloaca. At all events an operation for the relief of the ruptured perinæum cured the patient of the distressing complaint. But there are other cases where the vagina becomes filled with air from outside just as it does when a Sims' speculum is passed, and this air is then expelled with a noise in straining efforts, coughing, and the like. A vaginal plug or pessary will often remedy this.

CASE 8. *Menstruation during pregnancy and lactation.*—L. D—, æt. 32, married, complains of bearing-down pains and weakness and pain in the cardiac region. Menstruation has always been regular and she has had five children, with each child she has menstruated regularly both during the pregnancy and during the lactation ; so that she has never been able to calculate the age of the child *in utero*. An interesting point in the case, which she told spontaneously, is that she has a sister who does the same, and her mother always did the same during her pregnancies and lactations.

On examination the uterus was felt bimanually and did not present any abnormality beyond some anteversion. The sound passed the usual length (two inches and a half) and no evidence could be obtained of any bifid uterus or other solution to the above history. She had external hæmorrhoids which have bled latterly.

She is at present suckling her baby six months old, and her menses appear regularly.

CASE 9. *Procidentia uteri*; ulceration produced by cup and stem pessary.—I. C—, æt. 50, married twenty-two years. Has had one child twenty years ago, and ever since has had an aching pain in the left groin. For the last five weeks has had a watery and occasionally red discharge from the vagina. Attends now because her womb comes down. Has done a great deal of hard work.

On examination the uterus is found prolapsed so that the cervix with the inverted vagina protruded over two inches beyond the vulva. The sound passes nearly four inches; no erosion. A fortnight later a No. 3 ring pessary was placed in the vagina after the reduction of the prolapsed uterus, which was very easily effected. Meantime the patient was taking Mist. Ergotæ ℥j, t. d. A week after this she came and said that the ring pessary had only kept *in situ* one day and then it slipped out. A No. 4 was now put in, but this also slipped out, so she was fitted with a cup and stem pessary made of vulcanite. She was shown how to insert it and instructed to remove it every night and replace it every morning. For six weeks she went on very well so far as keeping up the uterus was concerned, but the last week or two had felt pain from the instrument.

On examination, the cervix being made to protrude through the vulva by voluntary straining, it was found that the rim of the cup of the pessary had ulcerated the mucous membrane on the outside of the cervix. The ulceration was annular like the rim itself but not perfectly continuous, and in some parts, chiefly in front, it was broader than in others. The largest patch was to the left and anteriorly. There was no ulceration anywhere else. She was told to rest in bed and go without the instrument for a week, after which ring pessaries were again resorted to until the ulceration was healed, but they failed to give much relief. The ulceration was treated by rest, glycerine, and cotton wool, and rapidly got well. She is still attending and a Zwanke's pessary is being tried.

RETROVERSION AND PROLAPSE.

Amongst the class of patients attending the hospital, this is one of the commonest conditions met with; and as has been

frequently pointed out by others the chief sufferers are those who do heavy work, such as charwomen, laundresses, general servants, &c.

In several instances a chronic bronchitis, or asthma, or phthisis, or other respiratory affection, or biliousness with retching and vomiting, has been the only cause discoverable; though it must be remembered that hard workers are often troubled with a cough, which aggravates their condition very considerably.

The mechanism of its production is sufficiently obvious. When the abdominal muscles and the diaphragm contract, which they do both in hard work (washing, &c.), and in the acts of coughing, vomiting, and straining, the pelvic diaphragm (which is formed by the levatores ani, coccygei, pyriformes, sphincter ani, sphincter vaginae, transversales perinaei, as well as the triangular ligaments and other fasciae) must also contract, or else the increased pressure within the abdomino-pelvic cavity will necessarily drive it down. The sphincter vaginae is very much weaker than the sphincter ani, and in fact the part of the pelvic diaphragm supported by this sphincter is the weakest spot in it. It is for this reason that the vaginal walls and, consequently, the uterus, descend more often than any other part of the pelvic contents.

If a patient begins to cough during a vaginal examination the anterior wall and, to a lesser degree, the posterior wall, always descends even in a healthy woman, but where there is prolapse the effect is much greater. The same thing is observed when the patient "bears down," which she is asked to do when one wishes to bring out the prolapse, and the amount, of prolapse; the bearing-down is precisely the same mechanical process as the cough, the diaphragm and abdominal muscles are contracted and the glottis is closed.

When a rectal examination is being made and a patient "bears down" or coughs, it is very noticeable that the rectal walls do not descend to anything like the same extent, the posterior wall scarcely at all, and the anterior wall only slightly, although the cervix uteri can be felt to descend freely over the posterior vaginal wall.

The bladder is forced down with the anterior wall much more frequently than the rectum with the posterior wall, so that

cystocele is a much more frequent complication of prolapsus uteri than rectocele.

This complaint has been treated in numerous ways, according to the amount of prolapse and the complication present.

In the first place, any source of straining, such as constipation or cough, has been treated by appropriate medicines. In several cases the patient has been induced to change a laborious occupation to a more sedentary one. Thus, a hard-worked general servant became a needle-woman, and in her case, although the cervix was within an inch of the vulva, she got very much better without any instrument, which would most certainly have become necessary in spite of an intact hymen, had she continued her heavy work. But in most cases the patient is obliged to work hard for a living, and cannot obtain more suitable employment. Speaking generally, my experience has been that prolapse and retroversion of slight degree were best treated by Hodge's pessary; prolapse of considerable degree with perhaps some cystocele or rectocele or both, by the watch-spring ring pessary covered by india-rubber; whilst in the worst cases the womb could only be kept up by a Zwanke's, or by a cup-and-stem, pessary. Air-ball pessaries are useful in very few cases.

The following cases will serve as examples:

CASE 10. *Slight prolapse and retroversion.*—F. C—, æt. 36. Has been married for fifteen years but has never been pregnant. Menstruation always regular but scanty and painful. Complains of pains in the hips, shooting down to the knees during the last three or four weeks. Also bearing-down pains. Formerly attended this hospital and had an instrument applied (Hodge's pessary?).

On vaginal examination, the uterus was found to be slightly prolapsed and distinctly retroverted. A Hodge's pessary was applied in such a way as to restore the uterus to its normal position. This relieved the pain and she now only comes every month or six weeks to have the pessary cleaned. On one occasion it was found upside down and she had had some feeling of discomfort. Otherwise she says she is very much more comfortable with the instrument than without.

CASE 11. *Considerable prolapse and retroversion without pro-*

trusion through the vulva.—M. I. C—, æt. 21, married two years ago, has been pregnant three times ; the first time she had a living child at the full term, the other two were aborted before the third month. The last miscarriage was two months ago, since then she has had constant bleeding from the womb and pain in the back, for which she has come up to the hospital.

On vaginal examination the cervix was felt low down, and when she coughed or strained it descended nearly to the vulva but not outside, and she herself had never noticed anything outside. The cervix was hard, and the os externum closed. The uterus felt bulky and heavy but was easily restored to its normal position. The anterior wall descended with it but the posterior only to a slight extent. Bowels constipated. Micturition frequent. A No. 3 watch-spring ring pessary was applied, and when she strained there was little if any descent. She has been wearing this instrument ever since and it has answered its purpose admirably. She is able to remove, clean, and re-apply it herself. The micturition is quite normal. The metrorrhagia was treated with ergot on the supposition of some subinvolution following the miscarriage, the bleeding soon stopped and has not recurred.

PROTRUSION OF CERVIX THROUGH VULVA (PROCIDENTIA) WITH RETROVERSION.

In all the cases of procidentia the condition was what has been termed "allongement," that is to say, the cervix uteri was stretched to a greater or less extent, and though in one case the hernial mass was nearly as large as a foetal head (this was operated on in the hospital) still even in that case, as in all the others, the fundus uteri was still within the pelvis.

CASE 12. *Procidentia ; frequent micturition.*—M. I—, æt. 40, married twenty years, has had five children and two miscarriages. Comes because of bearing-down pains and frequent micturition. Has had prolapse for five years but has never worn an instrument. Much foul discharge frequently tinged with blood.

On examination, cervix found projecting an inch or more through the vulva, os slightly eroded, uterus easily reduced. A Hodge's pessary applied. Following week the pessary removed because of the pain it occasioned. A No. 4 watch-spring ring pessary applied. She has been wearing this ever since, coming up occasionally to have it cleaned. When she bears down strongly the ring can just be seen by separating the labia. The micturition is much less frequent.

This symptom of frequent micturition is very common in all degrees of prolapse and is not unfrequently cured, or at all events much relieved, when the prolapse is reduced.

CASE 13. *Procidencia*.—S. S—, æt. 42, has had eight children and ten miscarriages. Complains of protrusion of the womb. Has never worn an instrument.

On examination the cervix projects an inch through the vulva, sound passes four inches and a half with concavity forwards and with handle well forwards. Vaginal walls very lax, especially the anterior, but no erosion nor ulceration. No. 3 ring pessary applied.

June 7th.—Ring slipped out the same night that it was applied. Air-ball pessary applied.

21st.—Pessary kept in two weeks; replaced.

July 12th.—Pessary again slipped out. Replaced and more distended. Attended several times, each time the air-ball having slipped out, so that on Nov. 29th a cup-and-stem vulcanite pessary was applied, and this has kept up the uterus without giving her pain. In this case not only was there great relaxation of the vaginal walls and heaviness of the uterus, but she had a laborious occupation (charwoman) and was subject to bilious attacks with violent retchings.

CASE 14. *Procidencia*.—L. C—, æt. 37, has had two children. The womb has come down ever since the last confinement two years ago. She had a ring pessary put in nine months ago, but it came out after a time and she has got much worse since.

On examination the cervix projects about three inches beyond the vulva. No erosion; quite easily reduced. Ring pessary applied. Attended weekly after this, each week having the pessary reapplied, as it would not remain *in situ*. Finally, a

Zwanke's pessary was fitted and she was instructed how to remove it and replace it herself, in order that she could take it out every night. It has answered very well and the patient has become pregnant.

RETROFLEXION, RETROVERSION, AND PROLAPSE.

Of this common condition there have been numerous examples.

The amount of each displacement has varied. Mostly the retroversion has been more marked than the retroflexion, whilst the prolapse has not been great in any case.

All these cases have occurred in women who have had one or more children.

The presence of retroflexion has been established in every case not only by digital examination but also by means of the sound, for no matter how certain one may feel that a mass felt in the posterior *cul-de-sac* is the fundus uteri, because of its shape, tenderness, size, and continuity with the cervix, it is only by finding that the sound passes the full length or nearly so with the concavity backwards, that the diagnosis is established.

In one or two instances the flexion and version have been reduced by digital pressure either per vaginam or per rectum or in both ways, the patient lying on the left side or being placed in the knee-elbow position.

But in the majority of cases the sound has been used and the uterus restored by a sort of *tour-de-maître*, the finger pushing out the fundus simultaneously. This is a much better and less painful procedure than the digital method alone, for it is obvious that the finger cannot press the fundus so far forwards as the sound is able to do, and as far as the suffering is concerned, anyone who will try the two methods will be readily convinced that digital pressure causes much more pain when used without the sound. The only case where digital pressure should alone be used is when a patient may possibly be pregnant. Thus a woman presented herself with bearing-down pains, and on examination the uterus was retroflexed, retroverted, and slightly prolapsed; she had not menstruated for six weeks, although she had previously been quite regular. It was impossible to dia-

gnose such an early pregnancy and yet one did not feel justified in passing the sound. The uterus was restored digitally and a Hodge's pessary was applied. It was some weeks before a well-fitting instrument was obtained, and meantime the courses came on again, proving that the fears of pregnancy had been groundless.

CASE 15.—A. S—, æt. 22, has been married three years, and has had one child two years ago. Complains of difficulty in defæcation, a yellowish vaginal discharge, and scalding on micturition.

On vaginal examination the cervix projects forwards and the fundus can be felt behind, with a distinct angle also behind; there is little or no prolapse. Attempts to reduce it by digital manipulation in the vagina and rectum gave her great pain, and failed to restore the natural position. A week later a sound was passed with the concavity backwards, and by gently turning it round, at the same time pushing the fundus with the finger in the vagina, it was completely reduced. A Hodge's pessary was now slipped over the sound and adjusted. She has worn this now five or six months, having it taken out and cleaned once a month, and it has kept the uterus in good position, defæcation being quite painless. Just lately, however, the instrument has given her more pain and a smaller one has been substituted.

AMENORRHŒA; MONTHLY EPISTAXIS.

CASE 16.—L. J—, æt. 30, married eight years, has had six children, of which two were stillborn. Three of the living children died when seven or eight weeks old.

Menstruation commenced at the age of fifteen, and has always been very regular every four weeks, both before and since marriage, and not accompanied by much pain. The last parturition was three years ago. Patient comes for "stoppage of monthly flow for the last six months." States that for the last three or four periods she has had epistaxis instead of the proper menstrual flow, but with the same feelings as during her ordinary periods. Bowels constipated. No pain with micturition. Has lost flesh and has got very pale.

Vaginal examination.—Cervix shortened, anterior lip nearly

flush with the vaginal wall, os admits tip of finger, and its margins feel rigid; uterus cannot be obtained bimanually, but no tumour can be felt in the posterior cul-de-sac. With the speculum the mucous membrane is seen to be quite healthy. Ordered Mist. Ferri Comp. ℥j, t. d., and Pil. Rhei Comp. gr. x, occasionally.

Three weeks later (August 18th, 1883) it was noted that her face, arms, and neck appeared bronzed, but no black spots could be found on the mucous membrane of the mouth, tongue, &c.

Oct. 18th.—No better. No menses yet. Has headache.

27th.—She says the amenorrhœa has now lasted nearly a year; the monthly epistaxis continues, and she has great headache. The skin and conjunctivæ are yellowish, but there is no bile pigment in the urine, no albumen, no cardiac bruit, and no retinal hæmorrhage.

Nov. 17th.—Yellowness more marked. No bile pigment in the urine. Liver enlarged, projecting three inches below the ribs.

The patient has not been since, but the above notes seem to be worth publication on account of the monthly epistaxis, associated with cessation of menstrual flow from the uterus. Whether vicarious menstruation be possible or not I do not know. Dr. Wilks has always taught us to be very sceptical in any such case.

This patient evidently had some liver trouble and slight jaundice, though we could never detect any bile pigment in the urine. Perhaps the epistaxis was due to the hepatic trouble and jaundice, though it is difficult to see why it should only occur once a month, and that this should occur at the monthly period without flow from the uterus, unless one allows that the menstrual molimen did affect it. Indeed, menstruation is no longer confined to a mere flow of blood, and epistaxis, hæmoptysis, hæmatemesis, &c., are no more instances of vicarious menstruation than is the bleeding from a cut finger. But the question remains whether it is not possible for bleeding which is one, if not the chief, factor in menstruation, to take place from other situations than the uterus, in other words, may not the *metrostaxis* of menstruation be performed vicariously by the nose, lungs, stomach, or by ulcers?

HYPOCHONDRIASIS

AND

HYPOCHONDRIACAL INSANITY.

BY GEORGE H. SAVAGE, M.D.

THE healthy mind should be more objective than subjective, but in most cases of insanity there is a morbid tendency to self-contemplation. The personal feeling becomes intensified, and the relationships of the individual to his surroundings are changed or lost. The "Ego," as alienists are in the habit of saying, becomes perverted. This change may show itself in innumerable ways, at one time the patient's thoughts being concentrated upon his moral nature; at another, the ideas being fixed upon some part of his body. We meet with every variety and every degree of hypochondriacal disturbance, and in the following paper I purpose describing some of the ways in which hypochondriasis shows itself in an asylum. Hypochondriacal persons most frequently remain simply hypochondriacal and do not become insane. Although in many cases there is a neurotic history when hypochondriasis occurs, yet hypochondriasis may occur in patients without any history of insanity among their relations. I believe from what I have seen that hypochondriacal parents may start insanity in their offspring. It is difficult to draw a line between melancholia and hypochondriasis; the

hypochondriac is intensely miserable in some cases, and a patient who begins with the belief that his inside is knotted may end in the belief that he is dead or has lost his soul. It is commonly said that in melancholia the patient has morbid feelings and in hypochondriasis morbid sensations; but here again everything must depend on the definition of sensation and feeling.

The hypochondriac has essentially perverted sensations and dwells upon them, but does not as a rule go so far in explaining the causes of his misery as does the melancholy person. Hypochondriasis, as I see it, must be considered as a serious neurosis consisting of a perversion of intellectual power due to morbid concentration of ideas on one or more morbid sensations. There is primarily a feeling of profound bodily illness which keeps the patient's attention fixed. The sensation may be defined or vague. It is associated probably with perverted nutrition somewhere, but whether this starts centrally or peripherally is doubtful, although I am inclined to think in many cases the first cause is some repeated local irritation; a kind of physical worry which without fairly exhausting, prevents refreshment by rest. The generally accepted causes are divided into the moral and physical; the moral being the worry of an irritated and morbidly trained conscience which has been led to look upon all that is organic or animal in the man as only to be suppressed, ignoring the fact that strength is shown in ruling and controlling rather than in suppressing. Quack medical literature is as disastrous as quack theological or philosophical teaching. It is rare probably for a single cause, such as the above, to produce insanity, other conditions of physical weakness being generally superadded. Hypochondriasis of the ordinary type is most frequently met with in elderly men, but as seen in an asylum it occurs in young men and women as well as in those of middle and old age of both sexes. Although in the majority of cases there is no doubt about the mental unsoundness of the patient, the intellect is but slightly impaired; memory is good, association of ideas normal, there is a difficulty of concentration of ideas upon subjects apart from the patient's morbid sensation and hence apparent feebleness of Will; yet this is not associated with real loss of mental power. The patient is often unstable and emotional, and likely to injure

himself or others in an impulsive way. He has, however, a perfect knowledge of what he is doing. The symptoms may remit in the older cases, and pass away completely in those who are young or of middle age. Those cases are specially favorable in which the menopause, gout, or some physical illness is at the root of the sensory disturbance; the majority of cases, however, become chronic. In the latter case the patient is worn out or passes into a condition of automatic misery which may last for years. It is rare to see hallucinations in these patients, and what may be called their delusions are their false interpretations of some bodily feeling. Thus they will say they have no brains, no bowels, or no testicles, merely thereby meaning that they have not the ordinary sensations in the regions of these viscera. The hypochondriac is generally fond enough of reasoning upon his case, but is altogether illogical and will assert and reassert that he alone has been affected in a certain way, and evidence fails to convince him to the contrary. Two patients in Bethlem at the same time said they were unnatural, the one believing his brain was changed and the other thinking she was dying; both said that if I could show there was blood in their bodies they would be satisfied, but the evidence of a prick followed by a drop of blood had no effect on either. In these cases the present feeling seems to override all past experience and memory, so that a doctor of considerable experience and attainments, becoming hypochondriacal, forgets or neglects his anatomical knowledge in the interpretation of his feelings, although he can see clearly the condition of his fellow patients.

I think it will be found convenient to divide the cases which I am describing according to the seat of the pain most complained of, one group of patients complaining of their heads, another of their abdominal viscera, and a third of their reproductive organs. I shall also refer to cases in which the complaints are more general, one patient being impressed with the idea that he is eaten up by syphilis, and another thinking that he is at the point of death. It is rare to have cases complaining of pains in the extremities, most of the hypochondriac complaints being referred to the middle line of the body, although I have met with a patient who described himself as having morbid feelings in every vein in his body. As to the group of

cases in which the general feelings are the most marked, there may also be met with special complaints of particular pains as well as the general feeling of discomfort. The following is a good example: Eliza M. C—, single, æt. 24, no insane relations, first attack; this has lasted nine months, and followed tropical fever in the West Indies, which was also complicated by chronic dysentery. The two diseases reduced her to a condition of extreme weakness, and at the same time she developed ideas that she was dying, and refused to take food. No evidence of any kind had the least effect in persuading her she might be mistaken, and all such attempts caused irritability and a tendency to mental explosions, which disturbed her and caused annoyance to those near her. Extreme physical weakness, with the most pallid aspect, associated with a complete want of occupation, persuasion that her last hours had come, summed up the points of this case; and as far as recovery is concerned, everything must depend upon the ability to get her nourished. She has been some months in Bethlem, and no treatment has so far done her any good. She has been fed regularly, not being allowed on any consideration to do without meals, although frequently force had to be used when persuasion failed; cod-liver oil, the syrups of the hypophosphites and the like were given in vain. The feeling which started as one of impending death is showing signs of passing into one of sin against God, and already she is accusing the doctor of sin in forcing her to eat.

In many younger patients, especially women, there are complaints of being “unnatural.” These cases are interesting, as they show the connection between melancholia and hypochondriasis. One set of patients, suffering from amenorrhœa, will tell you they are unnatural in body, that they have lost their bones, or passed their spinal columns by stool; others say that they are unnatural and incapable of affection. Both these conditions are usually associated with some ovarian disturbance.

Syphilitic hypochondriasis occurs mostly in men. A man of middle age suffering from dyspepsia and sleeplessness becomes restless and emotional; he has probably been leading a retired moral life; but he imagines he has contracted syphilis. It is a chance whether he believes he is an unpardoned sinner, or has a loathsome disorder; being possessed with the idea that

he has a contagious disease, he may either set to work to treat himself, passing from one quack to another, or he may shun all society, for fear of causing harm to others. Such a case was under my care in Bethlem, and for weeks I was in a difficulty about the nature of his mental state; he was apparently so sane. The first symptom I noticed was his tendency to wash himself; and here by the way I would call attention to the frequency with which young hypochondriacal patients wash themselves, especially if their hypochondriasis is of the sexual type. Following the tendency to wash himself and his underclothing, I noticed anxiety about his food, and the trouble he took to eat so as to allow no crumb to remain undestroyed. One day he ate half an apple and threw the rest from the window. As soon as he had done this he was seized with the most violent grief and dread lest any one should pick up and eat the rejected apple. Opposition to his seeking it caused the first outbreak of violence, and once the power of self-control was lost the whole case was cleared up; he then told me of his dread of syphilis and his fear lest he should communicate it to others. The hypochondriacal condition was succeeded by one of religious exaltation, and after months of excitement he died of phthisis. Similar vague and hypochondriacal feelings may be met with in the earlier stages of insanity, and there is a special variety of general paralysis of the insane in which hypochondriasis is the first noticed symptom. In such cases I have noticed a recognition of the mental symptoms and an appreciation of their gravity by a person suffering from early general paralysis. Simple hypochondriasis may arise as a result of some physical ailment; and as I have said there is more danger to society when the ailment is connected with the reproductive organs.

In a man of over forty, of strong vigorous family, with no neurotic tendency, who had suffered from syphilis which in a way had changed the life history, preventing him as it did from early marriage, and leading to rectal and urethral trouble, sleeplessness was a habit, and without any cause that could be detected, his general health failed, his digestion became impaired, he complained of constipation, and lost flesh. There was a restless craving for sleep; and the one characteristic of his condition was his inability to free himself from thoughts about his physical condition and dread lest he should

become insane. There were almost hourly variations in his condition, so that he would go to bed fairly comfortable, but would be unable to rest unless he took chloral or morphia, when he would pass a few hours in quiet, but not sleeping. Towards morning another dose of narcotic would give him sleep for a few hours. In the morning he did not wish to get up, not that he was sleepy, but that he was unwilling to do anything. Without appetite he would take some breakfast, and then for hours would lie about unless aroused by others, and even then he would do nothing that he could avoid. Some days he would be brighter than others, thinking that his troubles were at last at an end. Each evening, after dinner, he would be more cheerful and companionable; there were no signs of intellectual loss. The feeling of intense misery without any delusions whatever, the constant concentration of his thoughts upon himself and his condition, his complete inability to rouse himself to action and his sleeplessness are the chief features of his case. Change of air, change of scene, occupation associated with others, reduction of narcotics and a diet likely to produce increase of bodily weight, all tended to his restoration. There were, as is usual in such cases, periods of great improvement separated by periods of the most profound depression. Cases such as the one mentioned may occur most frequently in single men, and may follow local bodily disease or some vicious habit such as that of morphia or chloral taking, and in some old habits of sexual excess or masturbation may produce the same results. There may be in such cases impulsive excitement, so that a patient always talking about his insensible condition may impulsively injure those near him, or destroy property. As a rule such cases are not suicidal.

Hypochondriasis with noisy and violent expression.—Alfred W—, married, æt. 50, solicitor, no family history of insanity; he has been sober and industrious. This, the first attack, had lasted five months. No known cause was given, but I afterwards came to the conclusion that this man had been living a very exhausting and straining life in the endeavour to lead in society, in his profession, and in his home life. He at first became restless, then depressed and full of dreads about his health. He believed he was unable to walk, that his nose was growing smaller, that his

blood was all gone, and that he was being starved. He believed his nerves were dragging down his face on one side. He would only talk about the dwindling of his limbs and of his features, and protested that his penis was now gone to nothing, and that he was impotent: morphia was tried without any good effect. He was always in trouble; he struck the attendants who had to dress and undress him, he smashed windows and furniture, at the same time talking of his former position and of the lowness of his companions. The uncontrolled conceit of the parvenu mixed with the monotonous loquacity of the hypochondriac describes him well. He was kept at Bethlem twelve months, during which time he gained neither mental nor physical power, and we were very glad to pass him on to another asylum, as we considered him incurable. I have heard recently, six years since he left us, that he is in much the same condition.

In the next case, hypochondriasis of a general kind followed syphilis. John E—, single, æt. 29, medical man. Insanity on the maternal side. First attack, attributed to chloral and to syphilitic infection of a finger. Two months before admission he was dull and gradually became possessed with the idea that he was damned; but the chief morbid symptoms were a fixed idea that he was dying. He asked people to feel his pulse; he said it was stopping, and that he was turning blue in consequence; he was restless and excitedly tore his shirt and other clothes; he fancied his brain and fingers were congested. He said he never slept; he was treated with morphia with but little effect. He moaned and cried, and at times rushed about destroying any property which was in his way.

He remained in this same restless state, varying only in degree, for about five months, when he became more quiet, was sent on leave of absence and was discharged well. He told me afterwards that the only medicine that did him good was anti-syphilitic, but I believe it gave him mental rather than physical relief just at the time he needed something to confirm his improvement.

Hypochondriasis with tendency to violence.—Christopher W—, married, æt. 38, oil and colourman, with no insane relations, his father died of apoplexy, general health good. Predisposing cause said to be the swallowing a fish bone and being

much alarmed at the passage of a probang. He had also had a blow on the head twelve months before. He threatened the surgeon who had passed the probang. He was full of fancies, such as that a vessel on his brain had given way, that his throat, stomach, and intestines were one and all wrong, and that he had congestion of the brain. He had some "nervous" attacks of trembling which passed off leaving him much as before. He was under observation for twelve months and then was discharged uncured. He seemed in no way weaker in mind, but he had lost weight and was more distinctly melancholic. As so commonly happens there was a real physical excuse, if I may use the expression, for his first ideas, but the explanation was out of all proportion to the cause.

As to the sensations in the head among the insane, there are various complaints of pains within the skull, differing altogether from the complaints of headache made by ordinary patients; such symptoms are still more definitely present in those who are hypochondriacally insane. In cases to be described patients complained from morning till night of the loss of their brain, one thinking his brain had swollen, another that it had peeled, a third that it was dried up, and a fourth that it was starved of its blood supply. In these cases there is no loss of memory nor any loss in the association of ideas, but the monotony of their expressions and the irritating effect of their constant complaints are destructive to the peace and discipline of any ward.

Hypochondriasis with feeling of loss of brain.—G. J—, married, æt. 46, merchant, apoplexy and paralysis, but no insanity in parents. This is the first attack of insanity, and no cause could be given. At first he appeared to be getting weak and ill and unable to come to decisions, then he became dull. He was at first at a private asylum; he thought his brain was shut, and that he could not think. He had an aperient given him, and from that time he believed his brain was removed. From the time of his admission till his discharge a year after he never ceased to bemoan his lot. He would point out his pains for hours together, and if no one were near he would narrate them to imagined visitors. He slept well, ate well, and was fairly nourished, but still believed that he had but a brief time to

live, and would ask for his friends to be sent for. His memory was good and his reasoning faculties apart from his delusion were normal. He became excited, and on several occasions he struck his head against the wall. He also picked his forehead into a raw spot which we could not get healed in consequence of his constantly irritating it. Morphia was tried and for a time produced quiet, but the effect was only temporary; atropine failed also to relieve him. A seton was inserted and kept open at the back of his head, but no treatment availed. His urine contained sugar in small quantities, sp. gr. being 1032, 36 ounces a day. The patient wrote letters daily, and was at times obstinate. He may become violent, and his cure is not probable.

Hypochondriasis with ideas of change in brain.—John S. W—, single, æt. 45, surgeon, no insanity in the family, one brother died of phthisis. The patient had been a studious and steady lad; he worked hard at his profession and was also a good naturalist, and had many and varied tastes. He was musical, a chess player, and a fair linguist. He was distinctly conceited and intolerant of opposition. He gives as an explanation of all his trouble an act of self-abuse in which he prolonged the pleasure by some undescribed means, and which at once produced a change in the circulation in his skull. He has steadily got worse, and his whole lamentation is that he is misunderstood, and he is constantly trying to explain the alteration in his body. He says he has no blood and that if we tried to bleed him we should fail, but he would not consent to abide by a trial when we put his arm in the position for bleeding with the bandage applied in the ordinary way. He said at first that his was the unpardonable sin. But later he said less about this. He is ever telling us that his case is unique, and he has given me a detailed account of the adhesions which we shall find after death between the dura mater and the calvarium. He says his only place of peace is in bed, and he is indignant because I will not allow him to lie there and die. He says he never sleeps; by day he is constantly screaming and crying, holding his head between his hands. He can recall himself and play chess, the piano, or discuss scientific matters. He indignantly denies having the symptoms of hypochondriasis. He is very dirty and

negligent in dress and person. Although this patient has many false ideas and complains of many bodily ailments, yet the one he is always bemoaning is the condition of his brain. He asks you to feel his head, which he tells you is fiery hot at one time, and icy cold at another. He still says his brain never rests, that the dilatation of the veins within the skull is permanent; and that there are effusions in the ventricles and in the membranes. He asks strangers to listen to the creaking in his head. He says that narcotics have no effect on him, and that this proves his unnatural condition. I found that anything in the way of sleeping draughts irritated and annoyed him. Arsenic and similar tonics have had no effect, and I fear nothing medical or moral will be able to turn the tide of this man's ideas; they are still wearing a deeper and deeper channel and in the end will destroy him.

Hypochondriasis with brain sensation.—Jane A. E—, single, æt. 44, lady's maid; no insane relations; this is the second attack of insanity, the last being in 1862, from which she recovered. The cause was said to be anxiety about her eyesight; she became desponding and in four months had to be admitted into Bethlem. She then said her brain had "gone squash," and she feels the blood trickling down. She thinks it is water and not blood which now circulates in her skull. She wants someone to kill her. To her life is torture. She has slept badly and had no appetite, but took food fairly on admission. She was always ready to talk about the explosions and crackings, the rushings and burstings, which took place in her head, forcibly recalling the descriptions of the "way the water goes down at Lodore." She believed others could hear cracking going on inside her skull, and asked me to place my hand on the vertex to feel the heat. She improved somewhat and was more objective in her life, but at the end of twelve months she was discharged uncured, although I believe it is possible she may get well under less happy surroundings. She has the fact of a former recovery in her favour, and her period of life is also an element which makes one hopeful.

Many other similar cases have occurred to me, and as a rule they are incurable. These patients sometimes become quiet and may be manageable at home.

The second group of cases of insane hypochondriasis contains those who are troubled by insane feelings in their viscera, mostly of obstruction to the gastro-intestinal tract. This group is naturally divided into sub-groups according to the portion of the tract to which the obstruction is referred. In some the belief exists that the obstruction occurs in their throats, in others the fault is placed at the epigastrium, and in a third the anus is supposed to be closed. Clinically one meets with two general classes among patients of this group, one with large pendulous bellies and the other with doubly concave retracted abdomens.

First, the group referring their sensation to the throat. This is especially interesting from its connection with hysterical feelings. I have met nervous young men with permanent globus hystericus; they were young and emotional, suffering with a dread of death from suffocation. They were willing to have probangs and tubes passed to see the nature of the obstruction; and I should be glad to know how many find their way to the throat specialists. Comparatively few of these cases require to be sent to asylums, but occasionally they refuse all food, and are obliged to be placed under control.

The following is an excellent example of the symptoms as seen in these cases and is also an example of incurability:

Hypochondriasis with throat obstruction.—Alfred S. J—single, æt. 35, bank clerk, father insane. This is the first attack, which began three months before admission with despondency. He had been steady and industrious, leading too quiet and subjective a life; there is no known cause for this illness. He became possessed by the idea that he had cancer of his throat, and he said he wished he were dead. He professed to desire to die on the battle field, or to be poisoned, and like most such cases talked more of his suicidal designs than he should have done. The only thing he would talk about on admission was his throat, and he was always wanting each fresh visitor to examine it. We examined it and passed probangs, but effected no relief. Assafoetida and valerian were as useless as arsenic and iron. He believed his blood was wrong, and though I demonstrated its healthy condition by the microscope he was unconvinced. He became angry and threatening when

opposed, but we managed to get him to take food and exercise, and thus to get him into good general health. He was discharged relieved at the end of nine months, and I have frequently seen him since. He will do no work, as he is convinced that he is dying. He is growing fat, but this he said is not healthy fat, but diseased infiltration; and so though as able to work as before he is convinced that he cannot, and prefers to live on a small pension, in which way he will pass the rest of his life.

As a rule these cases do not get well unless the symptoms depend on adolescent conditions. If they occur in advancing age the prognosis is bad. Often young men with these symptoms, associated with exhaustion from masturbation, recover, the treatment being change of surroundings, avoidance as much as possible of medical treatment and intemperance; and I find it is better to avoid places where invalids and *malades imaginaires* congregate, such as hydropathic establishments and health resorts. Regular outdoor exercise, early rising, sea bathing, and in some cases a sea voyage may be useful. Stimulants should be given with the meals, and regular diet at not too long intervals is best. A light breakfast, a mid-day meal, little or no tea, and a plain neat supper between eight and nine. If medicine is given, iron and quinine, valerianate of zinc, and in some cases gold salts will be found useful tonics. Before leaving this group I would refer to a case in which this feeling of throat obstruction was recurrent, so that the patient, who had separate attacks of insanity at intervals of several years in each, had the same idea that his throat was stopped and that he should never be able to swallow again. The symptoms and the mode of thought were completely hypochondriacal, so that although he had suffered from exactly similar attacks before, yet in the third he said, "Oh, yes, the other attacks were delusions, but now I am sure it is true; no one ever was affected as I am; I shall die. Nothing can ever pass my throat again." Evidence by means of the stomach-pump or bougie is of no use, but it may be necessary to persevere with forced feeding to save the lives of such cases.

Obstruction about the epigastrium.—The majority of these

cases belong to this second group. They are, in fact, but exaggerations of ordinary hypochondriasis. Patients generally are men of middle age, but similar symptoms are complained of by women at the climacteric. The onset of the disorder is slow and insidious. Fits of depression with complaints of epigastric hollowness, are followed by restless uneasiness, the whole of the symptoms being ascribed to some error of diet; a patient in this stage gives up one article of diet after another, discovering that beer no longer suits him, or that all his troubles are due to tea. The anxiety about his condition and the irregular way in which he is taking nourishment tell upon his strength, and in the end he becomes so emaciated and nervous that there is considerable danger if any acute illness set in. Of course he may be suffering from gout or from impaired digestion, and relief of the general condition may come to him, but generally the symptoms increase till he is unable to work and would die of starvation, convinced that there was some real organic disease. Such patients may be dangerous to themselves or others, although rarely actively suicidal; they would passively starve; they are cowardly and weak of will; they will tell you that they wished they were dead as their lives are perfect hells—but they have no power to kill themselves.

Impulsively they will bruise or injure themselves, strike their friends or destroy property, just as a person with the toothache may kick a dog or thump a table. It must then be remembered that certain of these hypochondriacal cases may be dangerous to themselves or others, and are therefore to be looked upon as irresponsible. It is a question whether such cases are best treated in an asylum, their tendency to take in morbid ideas rendering them susceptible to nervous influence. The first case I give is that of a woman in whom the symptoms slowly and steadily developed till she became a chronic case of weak-mindedness with hypochondriacal expression.

Frances J—, single, æt. 52, lady, no insane relations, mother died of phthisis. This is the second attack of insanity, the first occurred in 1874, lasted eight months, and recovered. The present attack began ten weeks before admission, with excitement, talkativeness, depression, and refusal of food. She believed herself eternally lost, and that nothing ever passed through her,

and that her blood was turned to water. She said she was gradually filling up, and became excited at attempts to feed her. She was thin and sallow, but with no signs of abdominal disease. She was dirty in consequence of her delusion, and would not admit that *fæces* came from her; and this is not an exception. I have known a patient who declared positively that his clothes were fouled by others. There may be a kind of double consciousness such as occurred to a patient recently under my care, who asserted she felt someone outside her drag her about and this person made the mess in her room. No medicine or treatment effected any change in the patient. She asks to be put out of her misery. She is getting fat, and I think she will probably pass into a chronic state of insanity with the idea of obstruction as the one colouring her life. We meet now and then what I have called gastric automata; such was a man long resident at Bethlem who did nothing from morning till night but record the next meal and describe its nature and time.

The next case is very similar to that of F. J—, except that it occurred in a man and ended fatally. William H—, single, æt. 49, commercial traveller, three sisters have been insane, mother died of phthisis. This was the first attack of insanity; there were several causes contributing to this attack, a severe injury to his head years before, sunstroke in America, the illness of one sister and the death of another. The first symptoms were five months before admission and began with restlessness and wandering from home; he believed he was ruined; he fancied he had injured his friends and relations; he was sleepless; he called himself villain, hypocrite, and madman. He refused food saying, "As he had a short time to live, why waste it?" On admission he had the sallow, anxious, abdominal look, the dark muddy complexion and the pale pearly eye. He said he was going to die and that his head was made of wood. He had to be fed with the stomach-pump. He constantly bemoaned the state of his inside and the compulsion to take food. He said nothing had passed through him for months, and nothing would again. He was full of the idea that his food passed from his gullet into his vessels, and he complained of the tons of potatoes under his skin. He would

talk any length of time about his visceral symptoms, and give new and strange explanations, always ending with the hypochondriac's creed, "There never was such a case in the world before."

He lost weight steadily and sank and died, there being no return of reason, though he recognised his sister before his death. The brain weighed forty-two ounces and appeared much and generally wasted; there was great excess of fluid. There was a cavity in one lung, and old adhesions in both. There was an atheromatous patch in the aorta, vegetations on the aortic and mitral valves, and an embolic mass in one kidney. Other viscera healthy.

Besides obstruction we meet with every variety of uneasy feeling and extraordinary interpretation of it in an asylum, one patient believing that his food passes into a vast cloaca, while another believes that the food must pass under his skin or into his circulation; and I might fill my paper with the graphic descriptions given by patients of the way their food is disposed of. Some are specially fond of writing descriptions, and one went so far as to give me a graphic account, with plates, of a complete surgical operation for the restitution of his intestinal tract by the removal from a chloroformed pig of the stomach and intestines which were to be applied to the open end of his œsophagus. In his case temporary improvement was followed by fresh attacks, and in the end the patient became an inmate of one of the asylums for incurable patients. In the following case similar symptoms occurred in a young man. Frederick William M—, single, æt. 23, student of theology, his sister had suffered from melancholia. This was his first attack, due to overstudy and neglect of himself. The first thing noticed was that he had hallucinations of hearing which told him he was an angel; he thought he was dead and that electric sparks were developed in him. Soon after admission he objected to taking his food, and thought when he swallowed his food that it was stopped from entering him, and that when he went to the closet people tugged at his bowels. At one time he was convinced that all his food passed into his left arm, and he kept it across his chest to prevent it from escaping, and so for weeks he continued possessed chiefly by the ideas that he was directly communicating with his sister

and with other near relations, and that his food did not properly go into his inside. I believe in the end this case will recover. The prognosis in cases of hypochondriasis is practically the same whatever form the delusions assume, only it is not uncommon to meet with cases in an asylum in which, with all their complaints, there is a steady physical gain; the cases becoming weak-minded, but at the same time repeating in an automatic way the statement of their strange bodily condition.

In the next case hypochondriasis was the end of a case commencing with simple melancholia, the patient slowly developing ideas connected with uneasy abdominal sensations, and in the end dying exhausted with but few physical signs of disease.

Hypochondriasis following melancholia ending in death.—Frederick W—, married, æt. 49, clerk, grandfather insane from drink. First attack of three months' duration said to be due to over-exertion in business. He began with the dread of ruin; he said a voice told him it was better to kill himself and his children than starve. He began to refuse food, and said he could not take it. His abdomen was markedly concave; he had vomiting and constipation, but these passed off, though his bowels were, as a rule, very obstinate, and on two occasions the use of enemata produced alarming collapse. His bowels were not relieved for six weeks at a time, and while lying in bed he took little but a biscuit and a glass of brandy a day. He listened to what was said about him and was convinced he had got rid of all his abdominal viscera by the rectum. Month after month passed and the patient remained in much the same condition. No signs of physical disease appeared, but he had a sharp attack of diarrhoea. He very slowly sank and died. Post-mortem, a wasted brain and some old lung disease were all that was to be found.

In the next case the hypochondriasis was of a strangely recurrent type, so that the patient who one day was pleasant and amiable would on the next be most troublesome, violent, and destructive. That his symptoms depended upon some real gastric trouble seemed proved by the rapidity with which the symptoms would develop, the patient on several occasions becoming greatly disturbed within half-an-hour of a meal. All sorts of means were tried, from giving purges to administering

large quantities of oil; but nothing did any good, he had to be discharged uncured, and though tried with the greatest consideration at home it was found impossible to manage him out of an asylum.

Hypochondriasis with recurrent symptoms.—Edward C—, farmer, æt. 67, father died insane, brother weak-minded. First attack, following agricultural distress and money losses, had lasted three months before admission; it began with restlessness and sleeplessness; then he fancied his general health was failing, that his bowels were confined and that he should soon die. He would talk of nothing but his bowels; he hinted at suicide. His memory and general intellectual faculties were normal when aroused and in the intervals of his attacks. He was a well-nourished man with congested capillaries on his cheeks and a good-sized abdomen. Nothing abnormal could be made out. He had well-marked arcus senilis and some drawing to the right of the angle of the mouth. The chief interest in his case was due to the fact that he varied irregularly. Thus one day he would be happy and cheerful and say he should never have any of his queer fancies again, and he hoped I should let him go home; in a few hours, or in a day or two, generally soon after his mid-day meal, he would begin to cry and beg for his wife to be sent for before his death. He would declare that nothing would ever pass through him; he became more violent in his grief and would curse the doctors for not sending him home to die. As a rule he slept well and lost very little weight while in the hospital. At the expiration of fifteen months he was discharged uncured.

To complete the sketch of abdominal cases I must refer to those who believe that there is no passage in the lower part of the bowel. They resemble, in nearly every particular, those already described except that they are more troublesome from the fact that they are constantly attempting to effect a passage by introducing their fingers into the rectum. Local rectal treatment, warm baths, enemata may be tried, but it may become necessary to use strong gloves to prevent the patients injuring their bowels.

In the last group we meet with near allies to other forms of nervous disorder. Hysteria is often associated with this

nervous disorder; and ovarian disorder may also show itself in a special form of hypochondriasis. At the age when there are changes, whether of development or of decay, in the reproductive organs, morbid sensations may be insanely interpreted. And I shall have to call attention to the varieties one chiefly meets with.

Sexual hypochondriasis may occur in either men or women. It may occur in the single or married. It may occur in youth, middle, or advanced age. In men, the chief idea is that of impotence; in women unworthiness or feeling of being unnatural. The cases in which the feeling of being unnatural are described are connecting cases between melancholia and hypochondriasis. My reason for placing them among hypochondriacs is that they have morbid sensations due to some physical derangement which they exaggerate and misinterpret, but in which they still maintain complaints of bodily, and not so much mental or moral, defect. Thus, girls with amenorrhœa will tell you that they are unnatural; that they have no heart, no bones; that they are not human; that they are animals. I have never yet met with one saying she was sterile.

The young man who, either impressed that he has ruined himself by masturbation, or not comprehending the physical relationships of marriage, develops ideas that he is impotent, must be placed in this class. Such cases become dull, solitary, restless, impulsive, sleepless, much given to quack remedies, and are often distinctly suicidal. They complain of dull heavy feelings at the top of the head; sometimes the patients describe terrible feelings like aches or dulness in the brain. As I said, it may occur in young unmarried men, or in those who have been recently married. In the young single men I should suggest the possibility of a person suffering from continence as well as from incontinence or masturbation, and I am convinced that I have seen men kept perfectly ignorant of sexual matters by education and surroundings, who suffered from sexual hypochondriasis. I do not for a moment say that such cases would be cured by leaving the paths of virtue. I am in the habit of saying that at adolescence there is a struggle between the two kinds of love, the Eros and the Psyche, for the possession of the man, and in this struggle the

nutrition may suffer and mind may totter or fall, and the re-establishment must be slow and cannot be secured by any excess.

The examples of this group I shall divide into the younger cases of men and women, and the cases in advancing years in both sexes.

Among the younger men one meets with cases of so-called spermatorrhœa. As a rule these patients have been masturbators and have become possessed by the idea that they are losing all their mental power as well as their virility. I am not in a position to say what the condition of their reproductive organs is, but I do believe that the losses are mostly of prostatic fluid. Chas. A—, single, æt. 29, clerk, no insane relations, first attack of insanity. Earliest symptoms, complained of pains in the head and low spirits. He had been in the habit of masturbating from early boyhood. He was punctual, but indolent and inclined to alcoholic intemperance. Soon after the mental depression set in, he threatened suicide; he had recurring fits of intense grief associated with a constant dwelling upon the idea that his seed and strength were leaving him. He felt unable to control himself; he dreaded madness and was convinced he could never marry; he often had voluptuous dreams and complained of sinking pain at the epigastrium; and before admission he said that he had recurrences of uncontrolled lust and gave way to promiscuous intercourse; he had suffered from gonorrhœa. He was treated with phosphorus in ether and general tonic remedies, and was discharged recovered at the end of three months.

In the next case an older man was admitted for a second attack of insanity, which was similar to one that had occurred seven years before. Charles G—, married, æt. 43, father insane, previous attack said to be due to over-anxiety. On admission he was sure he had lost virile power; he was very suicidal, having several times tried to kill himself; he was full of the errors of his past life and was anxious for proofs of his marriage; he complained of seminal losses, and he thought that because most of his children were girls it was a proof that he was sexually weak. He was always dwelling upon sexual coitus and described in detail his manners of performing it with

the greatest anxiety as to one's opinion whether he was right or not. In the end he slowly recovered, and he attributed his recovery to a great extent to galvanism.

The next case is also one of a belief in impotence in a middle-aged man, who recovered, and whom after several years I know to have kept well and to have married.

Thomas W—, single, æt. 37, maternal uncle died in an asylum. This is the first attack, and began in December, 1879, and was said to have been caused by a love affair. My own belief is that the patient having contracted venereal diseases several times, and having generally looked upon women from the erotic side, was alarmed when he really became attached to a girl to find that his sexual desire lessened. He exaggerated the changed feelings, and fancied he was impotent, threw up his engagement, and this added sorrow completely upset his nervous balance. He was in another asylum till June, 1880, when he was brought to Bethlem. He was depressed and solitary, he was possessed with the idea that he was impotent and that he was accused of unnatural offences. He did not occupy himself in any way, but dwelt on his misery.

He could be roused and then spoke reasonably about horses, dogs, and his former work.

He lost flesh and for a time had to be forcibly fed. Within a few months of admission he recovered slightly for a time, but really he began to improve only eighteen months after admission. About this time I sent him out daily for drives with a job-master, who used to supply his master with horses, and who was willing to try the effect. Slowly he returned to his old habits and was discharged well at the end of May. Since then I have seen him; he is now married to his former sweetheart, and stands a fair prospect of keeping well.

I have already said that in women this idea assumes a different character, but I would say that the most common type is represented by the very curable class of cases in which the following is an outline of the history. A girl, generally of nervous stock, menstruates at the ordinary period for some year or two without any mental or physical perversion. Some moral or physical disturbance upsets her balance; in one case a disappointment in a love affair, in another a fright from a thunderstorm, or in

a third exposure to cold and wet during her menstrual period, is followed by arrest of menstruation.

The girl becomes nervous, sleepless, and sometimes chlorotic, she shuns her former companions, is given to tears, and if still engaged to be married she may throw up her engagement, or, what has occurred several times, when married she may decline sexual intercourse on the plea that she is not natural, that she is no longer a woman. This condition of things may follow a pregnancy and a natural delivery, so that a woman who up to the birth of the child has been healthy-minded believes now that she is unnatural. This feeling of being unnatural seems to be an extension of the feeling derived from amenorrhœa. In all these cases change of scene and exercise, stimulating diet, and the ordinary emmenagogues should be tried, although it frequently takes months for the re-establishment of the physical and mental health. In women about the climacteric, vague feelings of uneasiness connected with the reproductive organs may give rise to delusions of many kinds, the flushings of cold and heat which are described by nearly every woman at this period becoming more marked, or have a greater effect upon the neurotic individual.

Again, these cases may be divided into those in which the mental trouble exceeds the bodily, and those in which the bodily symptoms are chiefly felt. I have in this paper nothing to do with the miserable sinners whom we meet with in the first class, but would state that women at the climacteric will make the most astonishing statements as to ill-treatment which they suffer from. They dwell upon their bodily feelings and become convinced that persons rape them or injure them or their sexual organs; and it is of the utmost importance from a medico-legal point of view to recognise this type of insanity. They will tell the most matter-of-fact stories about persons injuring them, and will often complain of being rendered insensible before they are abused. They will show you trivial scratches, attributing them to their violators. Such patients may go on for years complaining of the same miserable feelings, and pass into weak-mindedness, still repeating their charges against men. Other cases of about the same age will complain of ammonia, chloroform, or some other irritant being applied to different parts of their bodies. In some cases local uterine

trouble may give rise to perverted feelings and cause hypochondriasis, which may be relieved by suitable local treatment.

One other group of cases may be referred to in which some foolish action, generally connected with sexual indulgence, has been done years before, forgotten, and condoned, but with some cause of physical weakness the memory of the folly or sin is vividly recalled, and the patient is utterly unable to throw off the feeling of wickedness or disgrace. Thus a patient will tell you that when a youth he exposed his person to his sister, or a married man will tell you he has never been able to remove from his mind the fact he contracted gonorrhœa while engaged to his present wife. Such patients will think of nothing but their supposed iniquity, and of the possible consequences to themselves, and others dwelling upon every sexual relationship, speak openly about it to anyone who will listen. These patients will generally recover if removed from their surroundings and occupied in some way out of themselves.

Already I feel that I have gone far enough in describing the chief phases in which uneasy bodily sensation causes absorption of all mental life around the acutely painful impression ; and I repeat what I said at the beginning, that it is after all but an arbitrary distinction to call certain cases melancholic and certain others hypochondriacal, unless one is able to look upon the first as primarily cerebral, and the second as only secondarily cerebral, being associated with disorder in the periphery.

NOTE OF TWO CASES
OF
GENERAL ANASARCA IN CHILDREN
WITHOUT ALBUMINURIA.

BY JAMES F. GOODHART, M.D.

MERRIS and Pepper, in the latest edition of their book upon the diseases of children, state that they have never met with dropsy after scarlatina in which they did not find albuminuria. Such has not been my experience. I have on several occasions in the out-patient room examined the urine of children suffering from slight dropsy and found it free from albumen. Many authors allude to such a condition, and as I believe that what may be called simple anasarca is not very uncommon in childhood, whether always in connection with scarlatina, or not, I do not know, I put short notes of the two following cases upon record.

CASE 1.—A boy, æt. 4, was in the Evelina Hospital in 1869, under the care of Dr. Hilton Fagge. There was no history of scarlatina, but he had been suddenly attacked, when in good health, a fortnight before his admission, with frequently recurring vomiting. He had been dropsical for five days, and when admitted was suffering from general anasarca, ascites, and some dulness at the base of one lung. The other viscera were normal. His urine was sp. gr. 1007 and contained no albumen.

Eight days later I noted that "no albumen has yet appeared in the urine; the dropsy is disappearing." He was still in the hospital three weeks later, but there is no further precise note

of him, but no doubt he left the hospital soon afterwards. Unfortunately at this distance of time I find my notes of the case to be very defective, but I know that I repeatedly examined the urine for albumen and always unsuccessfully.

CASE 2.—A girl, æt. $3\frac{1}{2}$, had scarlatina two months before she came into the Evelina Hospital. She was ill a fortnight, but was not kept in bed. Dropsy began in the legs a month before admission. The mother noticed that the urine was odorous, but it never altered its proper colour, and, according to her, it was abundant throughout. When admitted she was in a most remarkably anasarcaous condition, the whole of the subcutaneous tissue being affected, and the child looking quite like a distended bladder. The feet were blue and greatly swollen and she was in a cold and collapsed condition. There was no evidence of any desquamation. The small quantity of urine that was obtained contained no albumen. She was put at once into a wet pack, and this was renewed every six hours.

December 11.—The pack has produced no perspiration, but there has been abundant watering of the eyes. She has passed very little urine. The first sound of the heart is reduplicated, and there is a slight apex murmur. There is no increase of the præcordial dulness. The abdomen is tense; the left flank resonant, the right dull. The presence of fluid in the abdomen is doubtful. She is quite conscious. She has passed so little urine for two days that none could be collected.

12th.—The urine passed to-day is clear, sp. gr. 1015, no albumen, no excess of phosphates. The œdema is slightly less although still universal. The wet packs are being continued.

14th.—The urine to-day is very pale and clear; it contains a slight deposit of amorphous urates and a few uric acid crystals; no casts. The quantity cannot be estimated. The general œdema has diminished although still considerable. The face is pale and puffy and the feet the same. The abdomen is now lax; it contains no fluid. The liver comes an inch below the ribs; it is smooth and hard. The first sound of the heart is rough and reduplicated, but there is no murmur.

15th.—Slight dulness at right base. Pulse rather irregular.

18th.—The œdema has now almost disappeared from the legs and is much less elsewhere. Is passing plenty of urine; it is

free from albumen; the pulse is now regular and the heart sounds natural. The wet packs were discontinued.

21st.—Œdema rapidly disappearing from the whole body. Is passing plenty of urine; no albumen. Takes food well.

January 1.—The œdema is now entirely gone; passes plenty of urine; no albumen in it. Temperature normal. From this time she had no return of the dropsy. She was kept in bed for some days, taking iron for her anæmia. She left the hospital on January 22nd. The treatment consisted of milk diet, the wet pack, and an occasional jalap purge. Perchloride of iron was subsequently given for the anæmia which existed.

Steiner¹ writes of this affection thus: “Frerichs has described a rare form of dropsy without any disease of the kidneys, occurring after scarlatina, which he believes to be due to paralysis of the cutaneous nerves by exposure to cold during desquamation, and I have lately seen one such case where repeated examination of the urine revealed no change, whilst there was a very acute dropsy of the skin without any effusion into the cavities which lasted twelve days.”

Thomas² alludes to epidemics in which all the dropsical patients were free from albuminuria, and Hillier³ suggests that the slight œdema with which he alone has met may be due to anæmia, which is often very great and induced with great rapidity.

Perhaps Frerichs is warranted in looking elsewhere than the kidney for an explanation of these cases, but in the face of the well-known facts, to which I have drawn attention in a preceding article, and which seem to show that the anasarca of renal disease is independent of, although usually associated with, the occurrence of albuminuria, I think it is as well not too hastily to dismiss their renal origin. For if albuminuria is very often present without dropsy, and dropsy comes and goes without any distinct alterations in the course of the albuminuria, it is at any rate possible that the anasarca might in some cases be found as an isolated feature of some defective renal function.

Addendum.—I had written thus far when my attention was

¹ ‘Diseases of Children,’ Eng. trans., p. 341.

² ‘Ziemssen’s Encycl.,’ vol. 2, p. 259.

³ ‘Diseases of Children,’ p. 305.

called to a short article on a similar case by Dr. Dyce Duckworth in the recently published volume of the St. Bartholomew's Hospital Reports, vol. xix, 1888, entitled "Scarlatinal Dropsy with little or no Albuminuria." Dr. Duckworth alludes to many authors as having met with cases of this kind, including Gee, Dickinson, Bartels, and Henoch. In common with the authors he quotes, he inclines on the whole to regard such cases as occur after scarlatina as of nephritic origin.

It occurs to me to add that it seems not unlikely from the case recorded by Dr. Duckworth as well as from the second of those I have recorded, that the dropsy is in some way determined by a temporary suppression or diminution of the normal quantity of urine, a condition which, although usually followed by further evidences of inflammation and the appearance of albumen in the urine, may perhaps in occasional cases stop short of the more usual course.

ON ALBUMINURIA

AND

THE SYMPTOMS WHICH INDICATE ITS GRAVITY.

By F. A. MAHOMED, M.B. CANTAB.

IN a previous paper in these reports¹ I have discussed at some length the occurrence of Bright's disease without albuminuria, and the symptoms by which it may be recognised. In that paper it has been thought by some that I have too lightly treated the importance of albuminuria as a symptom of Bright's disease. It was very far from my intention to do so ; I desired to show that the absence of albumen from the urine did not necessarily preclude the diagnosis of Bright's disease. I now propose to inquire whether the presence of albumen in the urine necessarily indicates the existence of Bright's disease. While I do not wish to depreciate in any way the very great value of albuminuria as a symptom of disease, I am anxious that its value should be more justly appreciated, and that the importance of the presence or absence of other symptoms commonly associated with it in Bright's disease should be duly considered. In short, I desire to point out as far as I am able the symptoms which indicate the gravity of albuminuria. In order to do so it is first necessary that we should have some clear idea of the pathology of

¹ "Chronic Bright's Disease without Albuminuria," 'Guy's Hospital Reports,' vol. xxv, 1881.

albuminuria. The subject is one which presents many sides for our consideration, and concerning every side many contrary opinions are held. In such a matter it is well that we should occasionally reconsider our position with the view of ascertaining how far the opinions we may hold are in harmony with the most recent advances of our knowledge. Moreover, at the present time, though there may be little that is novel to say about its causation, there are several points concerning the recognition and significance of albuminuria that demand our careful consideration.

PART I.—THE EVIDENCE AFFORDED BY EXPERIMENTAL INVESTIGATIONS CONCERNING THE CAUSATION OF ALBUMINURIA.

Although a large amount of attention has been devoted to the experimental investigation of the pathology of albuminuria during recent years, our knowledge of it remains very incomplete, and owing to the contradictory results that have been recorded what little knowledge we possess appears uncertain. It will be unnecessary to review at length the extensive literature of the subject. I shall content myself with enumerating those experimental results which appear to me to have been well ascertained and concerning which the great majority of observers are agreed, leaving out of consideration those which are uncertain or controverted.

1. I take it as proved by a large number of observers, and generally accepted, that obstruction to the venous return of blood from the kidney by ligation or partial compression of the renal veins or of the vena cava above the entrance of the renal veins, produces albuminuria.

Senator appears to have demonstrated that the albumen, in these cases, comes from the tubular plexus. This he proved by rapid excision of the kidney and coagulation of the albumen *in situ*, after obstruction of the renal vein. He found that the albumen occupied the tubules, but did not appear in the capsules. If, however, the obstruction had been complete and prolonged, albumen was then found in the capsule also.

It is true that the inference that the albuminuria is produced by increased pressure in the renal vessels has been com-

bated by Runeberg, but his views on the whole subject appear to have been utterly distorted by the fallacious results of an elaborate series of experiments to which we shall refer hereafter.

2. It has been well established by Berzelius, Stockvis, Parkes, Pavy, Senator, and many others, including more recently Brunton and D'Arcy Power, that albuminuria may be produced by the presence of an abnormal form of albumen in the blood, such as egg albumen and peptones. This has been conveniently described as *hæmatogenous albuminuria*.

3. Nussbaum has taken advantage of the fact that while the renal artery supplies the glomerules of the kidney in the frog the tubular plexus receives its blood from another source, to perform some exceedingly ingenious experiments which prove that no albumen appears in the urine after injecting egg albumen or peptones into the blood of the frog when the renal arteries are ligatured and the blood supply to the glomerules is thus cut off, whereas albuminuria is invariably produced by injecting these readily diffusible albumens if the renal arteries are not ligatured.

These observations, taken in connection with the experiments of Ribbert, Posner, and Litten, to be mentioned hereafter, may be taken to prove that albuminuria usually results from transudation from the glomerules and not from the tubular plexus; though in acute inflammation of the kidney there is reason to believe that an inflammatory albuminous exudation may take place from these as from all other vessels.

4. The effect of the nervous system in regulating the blood supply of the kidney has been abundantly demonstrated. This may be brought about in various ways:

a. By the control exercised by the nervous system over the general blood pressure; its effect upon the kidneys will be considered hereafter.

b. A merely local effect can be produced on the kidney by stimulation or division of its vaso-motor nerves; or a contraction or dilatation of the renal vessels may be produced by a reflex stimulation or inhibition of their nerves.

c. It has recently been shown by Roy that the presence of certain salts in the blood circulating through the organs may produce a dilatation of the vessels and an engorgement of

the kidney after all the nerves connecting it with other parts have been divided. It is thought that this must be effected by the action of the salts upon some nervous structures within the organ itself.

5. The relation between the skin and the kidneys has received some attention from experimental pathologists, though not so much as it deserves. The results have been somewhat contradictory, but the injurious results of cutaneous asphyxia produced by varnishing an animal are well established, and albuminuria is usually one of them.

6. The experiments of Cohnheim show the part that disordered nutrition of the walls of the blood-vessels of the kidney may play in the production of albuminuria. After cutting off the blood supply from the kidney, by compressing the renal artery for a short period and then re-establishing the circulation, Cohnheim showed that the vessels not only became engorged with blood, but that exudation took place from the vessels into the surrounding tissues and into the tubes of the organ.

The same results have been obtained by Munk, Stockvis, and Hermann. Cohnheim's classical observations on the effect produced by inflammation in the blood-vessels of other organs may also be accepted as true of the kidney.

7. It has been suggested by Heidenhain and others that the epithelium covering the glomerular vessels may exercise a restraining influence and prevent the filtration of albumen into the glomerulus. This view has been elaborated by Dr. Coats in the recent discussion on albuminuria at Glasgow,¹ and he has put the case so well that it seems to me difficult to deny that the epithelium covering the glomerular capillaries prevents the exudation of albumen from the tuft under ordinary circumstances, and consequently that when the epithelium becomes injured by inflammatory action and deprived more or less of its vitality, it fails to perform its normal function, and, like dead epithelium, permits the exudation of albumen from the vessels. Dr. Coats draws a striking analogy between the epithelium of the renal glomeruli and that of the alveoli of the lung, and points out that when either is injured by inflammation, exudation of liquor sanguinis takes place.

¹ 'Trans. of the Glasgow Pathological and Clinical Society,' 1884.

8. The results of investigations into the part played by arterial pressure in producing albuminuria have been most contradictory. The experiments of Munk, Stockvis, and Hermann appeared to indicate that ligature of the renal arteries or compression of the abdominal aorta above their origin diminished the blood pressure in the kidneys and thus caused albuminuria, while ligature of other large arteries or compression of the abdominal aorta below the renal arteries was thought to raise the blood pressure, but did not cause albuminuria.

The fallacy underlying the first of these experiments has been demonstrated to be that ligature of the renal artery does not produce anæmia, but congestion and infarction of the kidney by means of the anastomotic circulation: this may occasionally be demonstrated in the post-mortem room as the result of embolism. In the second group of experiments the method employed does not increase the arterial pressure. While working at this subject experimentally in 1875 I satisfied myself of the inaccuracy of the deductions made from ligature of the abdominal aorta or large arteries. I found that the production of albuminuria in dogs was very easily effected, but its cause not so easily recognised; in fact, that the conditions of these apparently simple experiments were far too complicated to allow of any deductions being made from them; thus I found albuminuria often produced by the administration of chloroform and the ordinary operations necessary for observations on the blood pressure. I also found that ligature of the abdominal aorta produced scarcely any appreciable increase of arterial pressure. The effect produced upon the blood pressure of a dog by ligature of the abdominal aorta is shown in Fig. 1.

This experiment was performed on October 23rd, 1875, and the following notes were made during the operation:

11 a.m.—Dog chloroformed. Carotid exposed. Urine removed by means of a catheter contains an excess of uric acid. Slightly albuminous. No reaction with guaiacum test for blood.

11.10—Carotid connected with a mercurial manometer, recording a continuous trace on a revolving cylinder.

11.20.—Urine withdrawn. Contains much albumen and is

FIG. 1.

Aorta ligatured

perceptibly bloody. Blue reaction with guaiacum test. Immediately after this the blood pressure sank to zero; respirations stopped, animal moribund. By means of artificial respiration blood pressure rose slightly above the normal, and natural respiratory movements again commenced.

11.40.—Urine extremely bloody to the eye. Pulse irregular.

12.—The urine had lost all appearance of blood; it gave slight reaction with guaiacum test and was slightly albuminous.

12.25.—Urine gave the faintest green tinge with guaiacum test; it was slightly albuminous; of pale colour; no crystals of uric acid.

12.45.—Blood pressure falling. Urine bloody to the eye.

1.30—Urine still slightly bloody to the eye. Aorta tied below the renal arteries.

1.38.—Urine much more bloody. Heart's action slower. Pressure by manometer much the same.

1.40.—Urine less bloody, but more so than before ligature. The urine after ligature, though containing more blood colouring matter, did not appear to contain more albumen.

1.50.—The urine gave no reaction with guaiacum test and contained but little albumen. The quantity had increased.

The blood pressure was now steadily falling, and the dog died soon after from failure of the heart's action, the blood pressure having fallen to zero.

The results obtained during this experiment appear to show very clearly that the administration of chloroform is sufficient to produce albumen and blood in the urine of a dog; that these are most abundant when the respiration becomes embarrassed. The arterial pressure then falls and venous engorgement occurs. The albuminuria diminishes and the

FIG. 1.

.....

flow of urine increases when the blood pressure rises to the normal. On the other hand ligature of the abdominal aorta failed to raise the blood pressure to any appreciable extent, and we have, therefore, no evidence from this experiment whether an increase of blood pressure causes albuminuria or not.

I considered the conclusions from my experiments so unsatisfactory that I refrained from publishing them, thinking that the want of agreement with the results obtained by others was due to my own lack of skill in conducting the observations. More reliable results have since been obtained by other observers, and Professor Roy in his recent demonstration at the Royal Medico-Chirurgical Society of London especially dwelt upon the very great difficulty that physiologists have found in producing experimentally any variation in blood pressure in the lower animals. He showed that the ligature of any two large arteries or of the abdominal aorta, and also that the injection of a large quantity of defibrinated blood or salt solution, alike failed to raise the arterial pressure above the normal, though such methods might raise it to the normal if it had previously fallen below it.

One remarkable piece of evidence given by Michael Foster in his 'Text-book of Physiology' has not, I think, received sufficient attention. Dr. Foster in speaking of the influence of blood pressure on the secretion of urine, points out that the flow of water from the kidneys is merely a process of filtration from the glomerules, and that it is increased by raising the arterial pressure and decreased by lowering it. In the course of his observations he relates the following striking experimental results, which appear to me entirely in accordance with my own clinical observations on the pathology of Bright's disease and albuminuria. I will quote Dr. Foster's own words. After stating that section of the spinal cord below the

medulla brings about a great fall in blood pressure and diminution of the quantity of urine secreted, he goes on to say—"Stimulation of the spinal cord below the medulla, though acting in the converse direction, brings about the same result, arrest of the secretion. By the stimulation, the action of the vaso-motor nerves is augmented, and constriction of the renal arteries, as well as of other arteries in the body, is brought about. The increase of general blood pressure thus produced is insufficient to compensate for the increased resistance in the renal arteries; and as a consequence the flow of blood into the glomeruli is largely reduced. Indeed, on inspection the kidneys are seen during the stimulation to become pale and bloodless.

"Section of the renal nerves is followed by a most copious secretion, by what has been called hydruria or polyuria, the urine at the same time frequently becoming albuminous. The section of the nerves by interrupting the vaso-motor tracts, leads to dilatation of the renal arteries, and this to increased *capillary* pressure. If after section of the renal nerves the cord be divided below the medulla, the polyuria disappears; for the diminution of general blood pressure thus produced more than compensates for the special dilatation of the renal arteries. Conversely, if after section of the renal nerves the cord be stimulated, the flow of urine is still further increased, since the rise of general blood pressure due to the general arterial constriction caused by the stimulation tends to throw still more blood into the renal arteries, on which, owing to the division of their nerves, the spinal stimulation is powerless."

Concerning all experimental investigations of arterial pressure I must repeat what I have pointed out elsewhere, that physiologists are at present able to afford us very little reliable assistance in pathological inquiries of this nature, as they have no means of producing "delay" or "acceleration" in the capillary circulation throughout the body. They can only modify arterial pressure by regulating (1) the *inflow* or (2) the *capacity* of the arterial system; these results they can produce, the one by increasing or decreasing the frequency and the force of the cardiac contractions, the other by causing contraction or relaxation of the muscular coat of the arteries;

they are unable to alter the outflow by retarding or accelerating the capillary circulation, and there is good reason to believe that this is by far the most important factor in the pathological variations of arterial pressure. An inquiry whether there be conditions which on the one hand cause the blood corpuscles to linger in the capillaries and tend to cling to the walls, or on the other, let them pass rapidly through the vessels, is perhaps the most important that claims the attention of the physiologist. For my own part I should be interested to know more of the variations in tension of the gases in the blood and tissues. It may be that in the varying rapidity of the production and discharge of carbonic acid and the absorption of oxygen we may have the key to the control of the capillary circulation.

In connection with the experimental investigation of the pathology of albuminuria and the part played by arterial pressure in its production, I may observe that the subject has for some years been obscured by the unfortunate statements of Runeberg concerning the filtration of albumen. Drawing his conclusion from an elaborate series of experiments on the transudation of albumen through compound animal membranes under various pressures, Runeberg thought that he had proved that the generally accepted belief that *high* pressure promoted the filtration of albumen was a wrong one, and that *low* pressure was the essential condition. Having, as he thought, proved this experimentally, he proceeded to make his clinical facts suit his new theory, and succeeded in doing so by means of a variety of ingenious explanations and distortions. There is no doubt that Runeberg has caused great confusion and has much disguised the truth by these observations. Nevertheless, Medicine has derived some advantage from them, inasmuch as they have compelled attention to this matter and have themselves possibly revealed a certain amount of truth and explained what may happen under exceptional circumstances. The fallacies of Runeberg's observations have, however, been exposed and his criticisms fully answered by Heidenhain in Hermann's 'Physiology' (1880), and we need spend no further time on their consideration.

Since writing the above I have read the remarks of Professor Hamilton in the discussion on albuminuria at the Glasgow

Pathological and Clinical Society. His observations on the possible obstruction to the capillary circulation by the alteration in the specific gravity of the serum and its consequent mechanical effect upon the circulation of the red corpuscles are full of the deepest interest. I look forward most anxiously to a report of his promised experiments, but I am not prepared to accept as an hypothesis the statement that the specific gravity of the blood serum in Bright's disease is invariably reduced whenever the arterial pressure is increased. There appears some room for doubt in this matter; the statement is chiefly founded on the observations of Christison which were made many years ago, when the methods of observation were not so complete or so precise as at present. It is true that Bartels gives the specific gravity of the blood in five cases of extremely advanced Bright's disease, and that the specific gravity ranged from 1080·5 to 1021. He nevertheless declines to admit that there is any evidence of such an alteration of the blood in the early stages. On the contrary he speaks as follows—"But from the excellent state of nutrition and undiminished strength which so many individuals enjoy for years after their renal disease has begun, we must conclude that the blood preserves its normal properties for an equal length of time, and that its normal constituents are combined in their normal proportion in spite of the kidney affection." In support of the belief that there is no very great reduction usually present we may turn to the observations of Quincke;¹ in his table of twenty-two observations made on the blood of various patients, we find that the sp. gr. varies from 1062·1 in a case of typhoid fever, to 1035·2 in a case of chlorosis. His table includes five cases of Bright's disease, and the sp. gr. in these cases was found to be as follows:—1050·5, 1048·7, 1047·3, 1047·0, and 1041·1, the last observation having been made on a case of advanced renal cirrhosis. It is right to mention that there was only one case in the list below these, one of chlorosis, 1085·2; in fact there were only three other cases below 1050, one a case of chlorosis 1049·1, one of cirrhosis of the liver, 1049·6, and one of splenic leucocythæmia with a sp. gr. of 1044·3. So that here we find some confirmation of the statement that the specific gravity is reduced,

¹ 'Virch. Archiv,' vol. liv (1872), p. 537.

but on the other hand evidence that the reduction is not excessive; it must also be remembered that all of his other cases (except two of diabetes) were examples of *acute* diseases, while certainly three of the cases of Bright's disease were very *chronic*; there is no evidence how long the disease may have existed in the other two cases. In all cases of chronic disease deterioration of the blood is likely to occur.

There is yet another point I would submit to the consideration of Professor Hamilton; it is that I have elsewhere afforded evidence that changes of arterial pressure in slight cases of scarlatinal albuminuria can be produced with great rapidity, even in so short a time as an hour (by hot packs), and that the albuminuria coincidently disappears; it may be possible that the specific gravity of the blood is susceptible of equally sudden variations, but I am inclined to think that physiologists would not be prepared to admit this. I may further remark that the greatest increase of arterial pressure ever observed is found in the most acute cases of nephritis with suppression of the urine, and this may be produced within twenty-four, perhaps within twelve hours; while the lowest specific gravity observed in the blood has been in extremely chronic cases. Lastly, I may point out that I have demonstrated that the increase of arterial pressure precedes the appearance of any symptoms of renal disease; the urine has not diminished in quantity or increased in specific gravity, nor is there any evidence of the retention of fluid in the body by the occurrence of dropsy. I am led to make these criticisms of Professor Hamilton's theory, because I fear from its very attractive and apparently simple nature, his hearers and readers may be only too ready to accept and adopt it, before it has been fully proved. This I am sure its author would be the last to wish. I therefore venture to put forward these facts in support of a plea for further consideration of the matter.

**PART II.—CLINICAL AND ANATOMICAL OBSERVATIONS ON THE
PATHOLOGY OF ALBUMINURIA.**

Let us now turn to the teaching of clinical observation and morbid anatomy. From these we may draw inferences the truth of which we may be able to test by experiments upon the lower animals, but I should view with suspicion any doctrine of pathology that was based alone upon such experiments or upon attempts to imitate by physical means the complex conditions of the living body.

Since the glomerules are regarded by all as filters, we are justified in comparing the circumstances under which simple transudations take place in other parts of the body with the circumstances under which transudation takes place from the vessels of the kidney; and from the pathology of the various dropsies we shall find much which throws light on the pathology of albuminuria.

1. It is well known that obstruction to the venous return from a limb, whether by thrombosis of veins or heart disease produces an exudation of blood serum into the loose cellular tissue. Similar conditions are known to produce albuminuria. As examples I may mention the albuminuria of heart disease, that associated with dilatation of the right side of the heart secondary to lung disease, and the albuminuria occasionally produced by compression of the vena cava by new growth, as in Bartels' remarkable case of syphiloma of the liver compressing the vena cava. In these cases no structural alterations are perceptible in the kidney; unless they have existed for a long period; the organs may then become unduly tough and hard from increase of their fibroid tissue. In such conditions as these we find sufficient evidence that albuminuria is produced by the increased pressure upon the venous ends of the renal capillaries. It must be remembered that the element of increased venous pressure may greatly modify the urine characteristic of Bright's disease with granular kidneys. In consequence of the dilatation of their hearts cases of chronic Bright's disease may develop the dropsy, albuminuria, and scanty urine characteristic of heart disease; these symptoms must be dis-

tinguished in such cases from the dropsy and albuminuria of kidney disease and treated accordingly. Again, in the albuminuria of fever, and other exhausting conditions, and very probably in that of anæmia, increased venous pressure may be the determining cause; in these cases we have relaxed vessels that have lost their tone, associated with low arterial tension; the heart is weak and blood is apt to accumulate in the lungs and veins, the feeble heart lacking the power to drive it briskly through them.

While speaking of the albuminuria associated with fever, it is necessary that I should allude to the observations of Klebs,¹ Bouchard,² and others, which have been thought to prove that the albuminuria occurring in the course of the specific fevers is due to the presence of bacilli in the kidney; the condition has been described as "infective nephritis." I can conceive it possible that there may be some cases which merit this description; it might apply to those in which a definite nephritis, with bloody urine, occurs in cases of infectious disease, especially in enteric fever and diphtheria; but this view does not in the least explain the albuminuria usually associated with the prostration of prolonged fever from any cause, or with a severe degree of pyrexia.

I must also refer to the elaborate and ingenious experimental inquiry carried out by Dr. Walter Mendelson, of New York, "On the Renal Circulation during Fever," to which was awarded the Cartwright prize for 1888. Although these investigations were performed at the pathological institute of the University of Leipzig and under the auspices of Professor Cohnheim, the results are so astonishing that I feel it impossible to accept them until Dr. Mendelson's conclusions have been confirmed by further observations. According to his results it would appear that the condition of the kidney during fever is one of great anæmia due to the contraction of the renal arteries; the albuminuria of fever is ascribed to the bloodlessness of the organ and the consequent imperfect nutrition of the epithelium of the glomerules. Dr. Mendelson points out in his appendix the remarkable increase of arterial pressure which accompanies the increase of temperature.

¹ 'Arch. für Exper. Path.,' Band viii, ix, xii, xiii.

² 'Revue de Médecine,' 1881, p. 671.

His tracings certainly show a close relation between three conditions; as the temperature of the dog rises, the blood pressure invariably rises also, and the kidneys contract; the greater the elevation of the blood pressure, the greater the contraction of the kidneys. May not these last two conditions stand in the relation of cause and effect? Is it not highly probable that the renal arteries contract merely to protect the kidneys from the damaging effect of high arterial pressure upon their delicate tissues? In fact Dr. Mendelson actually quotes experiments by Roy and Cohnheim showing that "a contraction of the organ, consentaneous with the rise of the general arterial pressure, occurs when a peripheral stimulus is applied to any part of the body." Hence it appears to me that the anæmia of the kidneys which he has demonstrated in these cases of artificial fever is due not to the pyrexia, but to the increase of arterial pressure which was found to accompany it. I am not, however, prepared to admit that fever usually produces an increase of arterial pressure; as a rule, low arterial pressure prevails; but I have pointed out in one of my papers on the "Clinical Use of the Sphygmograph"¹ that in some cases arterial pressure is high notwithstanding the existence of pyrexia. I gave examples of this in cases of surgical fever or septic fever and in a case of severe contusion with profound shock. It would seem that the fever produced by Dr. Mendelson by the injection of pepsine in some of his cases, is likely enough to be a septic fever accompanied by high arterial pressure. His "thermic" fever produced by placing dogs in highly-heated chambers may resemble in its effects the "peripheral stimulus" mentioned above; moreover, it is likely that some degree of shock may be produced in dogs by destroying their *optic thalami*, a method employed in his cases of "peptic fever" to produce anæsthesia. Other causes of fallacy may exist in the various operations employed in these experiments.

2. The occurrence of various forms of albumen in the urine, owing to a diet of eggs, or in association with Bright's disease, or with dyspepsia, has been noticed by a large number of observers, including Parkes, Pavy, Claude Bernard, Lehmann, Edlefsen, and Senator; indeed, the last-mentioned

¹ 'Medical Times and Gazette,' Nov. 1, 1878.

physician maintains that peptones are normally present in every urine. How far the presence of peptones should be recognised under the term albuminuria is a matter for our discussion hereafter, but it appears probable that they may occur in the urine, and that they may largely increase the apparent quantity of the albumen lost in Bright's disease.

In connection with this question of abnormal forms of albumen in the blood, I may allude to a recent paper by Professor Semmola,¹ which appears to contain many important facts. Professor Semmola believes that the primary departure from health in cases of Bright's disease is caused by a failure to properly utilise the albuminoids of the economy. He believes that a healthy kidney can excrete albumen, but if this abnormal function is prolonged the kidneys become diseased. He has found the albuminoids of the blood in Bright's disease diffuse more readily than the albuminoids of the blood in other forms of albuminuria; and he thinks that albuminuria is due to the presence in the blood of albumens of greater diffusibility than the normal blood albumen. I shall hereafter adduce some facts which lend some support to this view.

8. The influence of the nervous system in the production of albuminuria is also undoubted and remarkable. Its parallel is seen in the dropsy of paralysed limbs, and in other circumstances indicating the influence of the nervous system in the production of dropsy. Professor Laycock,² who has written on neurotic dropsies, published a series of cases which appeared to him to indicate the close relation between albuminuria and nervous derangements; among others he quotes a case of intermittent hæmoglobinuria, as it is now called; anyone who has studied this interesting and remarkable disease must admit the very close connection between the nervous system and the production of albuminuria in these cases. Fürbringer³ has related some remarkable examples of the effects of fear and depressing emotions in the production of temporary albuminuria, and Sir Andrew Clark has recorded similar observations made upon students presenting themselves for the examination for Civil

¹ 'Le Progrès Méd.,' No. 24, 1883.

² 'Dublin Journal of Med. Science,' 1874.

³ "Zur Kenntniss der Albuminurie bei gesunden Nieren," 'Ztschr. f. Klin. Med.,' 1879, i, 340.

Service appointments. Although the nervous system is the exciting cause, the actual result is probably produced by increased arterial pressure brought about by general vascular spasm and possibly associated with a relaxed renal artery.

4. The intimate association between the skin, the bowels, and the kidneys seems almost too well known to need mention. The increased flow of urine associated with a cold skin, and the decreased flow which accompanies either a sweating skin or diarrhoea, are matters of daily observation. I have elsewhere demonstrated the remarkable and immediate effects produced by sweating and purging on the arterial pressure and on the albuminuria of scarlatinal convalescents.¹ Dr. O. Lassar² and Dr. Unna³ have both recorded cases of albuminuria following inunctions of the skin for scabies. In Dr. Lassar's case death occurred from oedema of the lungs, and the kidneys were examined microscopically and found to be perfectly normal. In Dr. Unna's cases the albuminuria was transient, and disappeared on the treatment being discontinued. I have repeatedly seen the association of temporary albuminuria with severe eczema, in cases in which the coexistence of gout and granular kidney appeared to be excluded. Professor Semmola, in the paper already referred to, has collected many cases in which albuminuria was associated with skin diseases. The albuminuria was cured in these cases by curing the skin disease.

5. Alterations in the tissue of the blood-vessels, tube-walls, and epithelium will be readily accepted as causes of albuminuria. It is in those cases of Bright's disease, either acute or subacute, in which the changes in these structures are most severe, that we have the most constant and most pronounced albuminuria. I have already referred to the important part which Dr. Coats considers that the glomerular epithelium plays in the prevention and production of albuminuria. We know that inflammatory changes in the vessels in other parts of the body give rise to the exudation of serum and even

¹ 'Trans. Royal Medico-Chirur. Soc.,' 1874.

² 'Virchow's Archiv,' Band lxxii, Heft 1, "Ueber den Zusammenhang von Hautödem und Albuminurie.

³ 'Virchow's Archiv,' Band lxxiv, Heft 8, "Albuminurie während der Styrax-einreibungen Krätzigers."

blood ; take, for example, the effusion of acute pleurisy. So that there can be no doubt that in these changes alone we have a sufficient cause for albuminuria.

While speaking of the relation of albuminuria to anatomical changes I should like to emphasise the following statements, which, I think, will be generally admitted. The more chronic changes in the kidney are confined to the fibroid thickening of vessels and intertubular material, while the epithelium is little if at all affected ; it is merely starved, ill-nourished epithelium. In these conditions very little albumen is to be found in the urine, commonly none at all. Such cases as these are liable to acute congestions of the kidney ; during these attacks albumen appears or increases in the urine, and epithelial and other changes occur in the kidneys. I do not say that the epithelial proliferation produces the albuminuria ; far from it, I regard them as two effects produced by the same cause, namely, inflammatory congestion. Whenever this occurs, whether in the lungs or in the gastrointestinal canal, it produces increased cell growth and epithelial proliferation, whereas on membranes such as the pleura it produces exudation of serum. In the kidney we have structures analogous to both, the glomerules readily permitting of exudations, the tubes lined by active epithelium always ready to proliferate on slight provocation. Another structural change on which great stress is often laid deserves special mention : I mean the changes described as "glomerular nephritis." From the examination of a large number of kidneys, scarlatinal and other, I have satisfied myself on two points : first, that the so-called glomerular nephritis is not a disease limited to scarlatinal cases ; secondly, that the amount of acute changes in the glomeruli is a fair gauge of the acuteness and severity of the attack. I believe that these cases are especially characterised by suppression of urine ; they are the rapidly fatal cases more commonly seen in connection with scarlatina than in any other condition, though occasionally arising from chill or other causes. It is the sudden and often extreme rise of blood pressure in these scarlatinal cases which throws such great strain on the glomerules ; the whole weight of the increased blood pressure falls upon them, for the renal congestion is accompanied by dilatation of the arteries and

intense hyperæmia of the kidneys. Though it may be true that these changes are more commonly found in scarlatinal cases than in others, it is certain that by no means all cases of scarlatina with albuminuria have unusually severe glomerular changes; on the other hand, the cases with these changes are comparatively rare in my experience. They are the severe and fatal cases of scarlatinal dropsy which we now see very rarely at the London Fever Hospital, owing to the exceedingly careful watch that is kept for early symptoms and to the very active and immediate treatment that is employed directly these symptoms appear.

6. There still remains for consideration the condition which I believe to be the most important factor in the causation of albuminuria, namely, increased arterial pressure. It is not, however, an absolutely necessary and constant one, for there are three other conditions which have been mentioned, each of which appears sufficient to cause it—increased venous pressure, the presence of easily diffusible albumens in the blood, and changes in the renal epithelium. I am content to accept the dictum of Bartels, which he lays down in his admirable article in ‘Ziemssen’ as follows:

“The outflow (passage) of serum albumen from the blood-vessels of the renal tubes of the kidneys will in every instance be proportioned either to an existing abnormal increase of the blood pressure, or to an altered state of the walls of the vessels, or to a combination of both these causes acting together.”

Bartels arrived at this conclusion before the very close relationship between high arterial pressure and albuminuria had been ascertained as fully as we now know it. I need not repeat the evidence I have elsewhere adduced in support of it. I may, however, venture to remark that the facts I brought forward ten years ago¹ have never been controverted, while on the other hand additional evidence has been constantly accumulating in support of them. I then showed that a great increase of arterial pressure *preceded* the appearance of albumen in the urine and was in fact the earliest symptom of Bright’s disease; that this was true not only of the acute disease as seen after scarlatina, but also in the chronic and

¹ ‘Trans. Roy. Med. and Chir. Soc.,’ 1874.

insidious forms of the malady. I further demonstrated that a similar increase of arterial pressure was exceedingly apt to occur during pregnancy, and that it explained both the albuminuria and convulsions associated with this condition. These latter observations were repeated and fully confirmed by Dr. Fancourt Barnes.¹ I also proved that in the early stages of scarlatinal nephritis the arterial pressure could be reduced in a remarkable manner by such remedies as the hot pack and purgatives, and I was able to show the immediate disappearance of albumen from the urine coincidently with this reduction of arterial pressure.

So far we have only regarded increased venous or arterial pressure as conditions which appear to favour the occurrence of albuminuria. We have yet to consider more exactly why they do so. The question has often been raised, why is not the urine albuminous in health? This question is especially put by those who believe that albumen normally transudes from the glomerules into the Malpighian capsules and is either reabsorbed by the cells of the capsules or by those of the tubules. The belief of Rosenstein and others, that albuminuria is caused by the failure of the epithelium of the tubules to reabsorb the albumen that has been poured out by the glomerules and which may be normally present in the renal tubules has recently received additional support by the observations of Dr. Newman, detailed at the Glasgow discussion. Concerning these observations I can only say that although Dr. Newman deduces some support from the minute anatomy of the kidney, his contention cannot, in my opinion, be regarded as proved by facts; on the other hand this hypothesis appears to me to charge nature with a very clumsy arrangement, quite unlike her usual simple and labour-saving methods. Moreover, evidence exists that albumen does not normally exist in the glomerules or tubules. Experiments have been made by Ribbert, Posner, and Litten which prove this, and are accepted as sufficient by Charcot in his lectures on 'Albuminuria.' These observers found an amorphous coagulum in the capsules and tubules of kidneys secreting albuminous urine after ligature of the renal artery; while in control experiments with healthy kidneys, they found the

¹ 'Obst. Soc. Trans.,' 1875.

capsules and tubules free from any such coagulum. These observations were made by rapidly removing the kidneys, and plunging them in boiling water to coagulate the albumen.

But more important than such experiments are the observations of Heidenhain and the facts adduced by Dr. Coats in the paper already referred to, which go far to prove that the healthy glomerular epithelium and its basement membrane exercise a restraining influence on the exudation of albumen, just as the epithelium and basement membrane prevent exudation of albuminous fluids in other parts of the body, where they cover delicate blood-vessels, as, for instance, in the alveoli of the lung. These authors regard the flow of water from the glomerules more as a secretion than a filtration, and they say that while a healthy epithelium restrains the exudation of albumen, a disordered epithelium will permit of its escape.

In considering the transudation of albumen from the glomerules there is one point to which attention has not been sufficiently directed. It is that the rapidity of the blood-flow in the glomerules is probably greater than in any other capillaries of the body; the glomerular arteries come off from comparatively large vessels; they are very short and immediately split up into capillaries which are larger than any others in the body; so large are they and so directly in connection with the arteries that I imagine that pulsation must actually occur in them. It seems probable that the more rapid the blood-stream the less likely is exudation of albumen to occur from the vessels.

So long ago as 1852 Dr. George Johnson in the first edition of his admirable work on "*Diseases of the Kidneys*" sums up the evidence of the causation of albuminuria in favour of its being due to an exudation from the Malpighian glomerules owing to an obstruction in the flow through the tubular plexus, produced either by the compression of the plexus by the tubules distended with epithelium, or to the compression or obliteration of the plexus by the formation of fibroid tissue between the tubules of the cirrhotic kidney. This view of albuminuria being due to increased pressure in the glomerular vessels is that held by Bartels, Charcot, and many other great pathologists who have devoted their attention to the subject, and the same view has been recently supported by Dr.

Newman, Professor Hamilton, and Dr. Coats in the discussion already alluded to. I believe that we may almost consider this point established. Charcot assigns the production of albuminuria to delay in the vessels of the glomerulus, but I cannot agree with his views as expressed in the following summary, which I quote in his own words:

“Thus in every case in which the disturbance of the general or local circulation determines albuminuria, it is, I repeat, neither the increase nor the decrease of the intra-glomerular blood pressure which is at fault; it is the retardation of the blood current, and consequently the prolonged sojourn of feebly oxidised blood in the renal capillaries. We thus find here again those conditions of anoxhæmia of the epithelial cells of the glomerulus which physiology has taught us to regard as unfavorable to the secretion of urinary water. These are the same conditions which preside over the secretion of albumen, and this circumstance explains the remarkable fact that in the albuminuria allied to circulatory troubles the urine is scanty at the same time as it is albuminous.”¹

I cannot agree with the statement that “it is neither the increase nor the decrease of the intra-glomerular blood pressure which is at fault,” nor do I know anything about the blood being necessarily feebly oxidised in albuminuria, nor of the condition of “anoxhæmia of the epithelial cells of the glomerulus.” What I believe is this, that the mere rapidity of the blood-flow through the glomerular vessels, while it permits of the exudation of water, does not permit of the exudation of albumen; that increased arterial pressure very severely affects the glomerules of the kidney, producing in them, when in a normal condition, not only a great increase of pressure, but also a great distension of their very elastic walls; that more blood will be poured into them than can be readily carried away by the capillary plexus, and consequently the rapidity of the blood-stream will be delayed; these conditions of distension and delay in the glomerules when combined are sufficient to produce a more or less severe albuminuria; but not necessarily any diminution in the quantity of urine. If in addition to this there is inflammatory proliferation of the epithelium of

¹ Quoted from Dr. Saundby's summary of Prof. Charcot's lectures, 'Lond. Med. Record,' vol. ix, 1881.

the tubes the urine may be diminished and made more albuminous.

This change in the quality of the urine has been attributed to certain anatomical conditions by Dr. Johnson, as mentioned above. The diminution in the quantity of the urine is probably caused by the obstruction to the flow of fluid from the tubes, caused by the increased proliferation of the epithelium and consequent plugging of many tubes; this also increases the pressure in the tubes and so offers increased resistance to the exudation of water from the vessels; the diminished flow may also depend in part upon the inflammatory changes in the Malpighian capsules, which if severe produce complete suppression of urine. The increased quantity of albumen is thought by him to depend upon the increased pressure in the glomerules; this is brought about by the compression of the tubular plexus by the tubules distended with epithelium, the resistance to the passage of blood through the tubular plexus causing obstruction to the flow in the efferent arteries and consequent increase of pressure in the glomerular vessels.

All these changes will be accentuated by the fact that they are accompanied by inflammatory hyperæmia of the organ, which is liable to be peculiarly severe on account of its vessels being relaxed while the general blood pressure is greatly increased.

In the chronic form of the disease, with granular kidneys, I believe that the effect of the high arterial pressure is less felt in the kidney than under ordinary circumstances, inasmuch as the glomerular vessels have gradually thickened and lost much of their elasticity, thereby adapting themselves to the increased pressure; while the Malpighian capsules have thickened and protect the glomerules from over-distension. No doubt great additional strain is thrown upon the glomerules owing to the compression of the tubular plexus by the development of the intertubular fibroid tissue, and this is often sufficient to produce chronic albuminuria; these chronic cases are also liable to frequently recurring acute congestions of the kidney, which, together with temporary elevations of arterial pressure, will account for the variable amount of albumen found in the urine.

I shall hereafter offer evidence which appears to indicate that in some cases more diffusible forms of albumen, such as paraglobulin and peptones appear in the urine before true serum albumen; they no doubt indicate early and less severe conditions of disturbance of the glomerular circulation.

From the tubular plexus we should not expect albumen to transude, for the structure of the tubules more resembles that of a mucous membrane and is well protected from mere exudation.

Concerning the exudation of the solids by the kidney, I am inclined to follow Dr. Johnson's view expressed so long ago as 1852, and confirmed in recent years by the observations of Heidenhain, that they are secreted by the cells of the tubules just as the bile is secreted by the liver cells; in the glomerules of the kidney we have an additional arrangement for filtering off a large quantity of water.

To sum up these conditions, which we may safely consider to have been demonstrated, both by clinical and anatomical observations and by experimental research, as conducive to albuminuria, we may state them as follows:

1. Increased glomerular pressure and slowing of the blood-stream in the glomerules:

a. From general increase of arterial pressure with local hyperæmia of the kidney, due to disorder of other excretory organs, especially of the skin and bowels. This may occur as a functional condition.

b. From general increase of arterial pressure, aided by obstruction of the tubular plexus, which may be compressed either by tubules distended by epithelium or by growth of intertubular fibroid tissue. This condition is necessarily due to organic changes.

c. From increase of glomerular pressure due to disturbance of the renal circulation through the nervous system, which may produce either a local engorgement or a general increase of arterial pressure. This may be purely functional, or may accompany organic changes.

d. From obstruction to the return of the venous blood. This may be due to a functional condition or to organic changes in other organs.

2. From alteration in the tissue of the blood-vessels, tube-

walls, and epithelium, produced by acute inflammation, infarction, or other less severe conditions.

8. From chemical changes in the blood, especially the presence of abnormal forms of albumen in the blood-serum.

PART III.—THE RECOGNITION OF ALBUMINURIA.

Having passed briefly in review the information at our disposal on which we may form some opinion on the pathology of albuminuria, I now proceed to that part of the subject which seems at present most urgently to demand our consideration.

Before we can discuss albuminuria, it is first necessary that we should clearly understand what we mean by the term. Hitherto it has been usually taken to imply the occurrence of the albumen of blood-serum in the urine as indicated by a precipitate produced by heat and nitric acid. Of late years the tendency has been to greatly extend the meaning of the term, first by allowing it to include the presence of a variety of other forms of albumen in the urine, secondly, by the introduction of numerous and more delicate tests. Many, no doubt, are content to accept the old definition and do not wish to see it extended, and they will urge that by such an extension the symptom loses its old significance and clinical value. With such I have much sympathy, but I am inclined to think that we cannot accept it for many weighty reasons, to which I shall have hereafter to allude.

In considering the subject it is necessary to remember the great complexity of the group of substances with which we are dealing and also their very unstable nature. Anyone who devotes much attention to the chemistry of the albumens cannot but be deeply impressed with the confused state of our knowledge and the great uncertainty attending the recognition of the various forms of proteids that occur in the urine. I shall not attempt to give an account of these various forms; those who desire information on the subject will find an admirable summary in Dr. Saundby's article on "Albumi-

nuria,"¹ and still fuller details in Bartels' article in 'Ziensen's Encyclopædia' and in Peabody's 'Supplement' to the same, bringing the information down to all that was known in 1881.

It is necessary, however, to allude to the albuminous substances which are thought to have been identified in the urine. Serum albumen is, of course, the chief of these, and is the one which we are commonly supposed to recognise, but belonging to the same class are the globulins, especially paraglobulin, which, according to J. C. Lehmann, Edlefsen, and Senator, is always present in albuminous urine. It is probable that this latter body is occasionally present when ordinary serum albumen is absent.

Next in order of importance are the so-called acid albumens and alkali albumens, but it is well to remember that each of these may include a whole group of substances—Foster calls them *albuminates*; amongst others may be mentioned the muscle albumen, which may appear in an acid solution as syntonin and in an alkaline solution as myosin; neither of these can be differentiated from what are called acid albumens, or alkali albumens, respectively. It requires but a little experience with these bodies to find how easily they may be overlooked by merely boiling the solution. Of course if the one is in an acid solution, the other in an alkaline, they will not be precipitated by heat till their respective solutions are neutralised, but if the process of neutralisation be carried too far they will exchange characters and still remain unprecipitated. As the acidity and alkalinity of the urine vary greatly, it is easy to understand that by the test of heat alone we are exceedingly likely to be misled as to the absence of these bodies.

Certain food albumens, generally classed as peptones, have been observed in the urine by Berzelius, Stockvis, Claude Bernard, Parkes, Pavy, Senator, Brunton and D'Arcy Power, Balfe, and many others. This group contains many varieties; they have the common property of not being precipitated by heat, and indeed when precipitated by other reagents they are dissolved by heat. Peptones are stated by many observers to be present in all albuminous urines to a greater or less degree.

¹ 'Birmingham Med. Review,' July, 1879.

In addition to these Gerhardt, Bence-Jones, Baylon, and John Greene have all described anomalous forms of albumen as occurring in the urine, to which the terms of hemi-albumose, met-albumen, leth-albumen, and albuminose have been applied with more or less accuracy.

I cannot leave this part of my subject without referring to the valuable researches of Dr. Kirk, of Glasgow, on the albumen present in the urine. His observations fully establish the difficulty of obtaining precipitates of albumen in acid and alkaline solutions by the aid of heat alone. Unfortunately he would add another to our list under the name of ren-albumen.¹

To my mind, it seems clear that we have to deal with many forms of albumen in the urine and not with *one* form, and moreover that these forms are probably constantly changing their characters from the very moment of their secretion by the kidneys until many days afterwards. I have no doubt that they undergo change in the bladder before they are passed, and I am certain that they undergo change in the urine glass after they have been passed, for I have repeatedly observed it; in fact, if one is comparing the reactions of the same urine with many tests, as I have been doing recently, it is by no means unfrequent to obtain different reactions after the urine has cooled from those obtained immediately it is passed, and again to obtain different reactions on the second day from those obtained on the first. This change in the character of the albumen is altogether apart from mere decomposition and the results of alkalinity, although it may indicate steps in that direction. It may be produced by the action of the salts or of the urea upon the albumen, or it may be due to micro-organisms. The whole subject is too complex and long to enter into on the present occasion, though I hope to do so at some future time.

Since writing the above I have discovered that a similar experience has been recorded by Sir Andrew Clark. He makes the following observation:—"A specimen of urine examined within an hour after extrusion from the bladder may yield unequivocal evidence of the presence of albumen, and cease to do so after twelve hours."²

¹ 'Glasgow Med. Journal,' May, 1881.

² 'The London Hospital Reports,' vol. i, 1864.

For these and other reasons yet to be mentioned, it appears to me to be wrong to limit ourselves to the use of heat and nitric acid, which are known to fail to give reactions with many of these forms of albumen (for instance, the peptones), and are moreover not so delicate as other tests recently introduced.

Concerning the whole of the new and more delicate tests for albumen, I believe that I best indicate their especial value by saying that *they are excellent negative tests*. By this I mean that if one or more of them is applied to a specimen of urine and no precipitate is obtained, we may safely conclude that the urine contains no albumen. Their errors are not those of omission, but of commission; they bring down many precipitates which are not albumen. Having obtained a precipitate by any one of them, it will be necessary to confirm the observation by other tests to ascertain whether or not it is really albumen, or whether the precipitate is due to some other cause. I am strongly of opinion that scarcely any one of them can be relied upon alone to establish a diagnosis of albuminuria, though any one of them may detect albumen when heat and nitric acid fail to do so.

It will perhaps be useful if I briefly mention what I believe to be the advantages and fallacies of the tests that I have employed.

I need not refer in detail to the fallacies of heat and nitric acid; they are sufficiently well known, but I would say with regard to these tests that there are forms of albumen which appear in the urine which elude sometimes the one, sometimes the other, and sometimes both. For example, my clinical clerk, Mr. Harris, who kindly undertook to make some special observations on the urine for me, called my attention on April 3rd, 1883, to the urine of a woman under my care suffering from anæmia. The urine was clear, of acid reaction, and of sp. gr. 1008. Nitric acid, used by Heller's method, gave no cloud; when acidulated slightly with acetic acid and boiled a thick cloud was obtained; there was a slight precipitate with picric acid and citric acid, a dense precipitate with mercuric chloride and with tincture of galls. There was also a dense cloud with heat when nitric acid had been previously added. There can, I think, be no doubt that this urine contained

albumen and that nitric acid failed to detect it. The condition lasted for about three days and then disappeared. I quote the case because the precipitates obtained were remarkably dense, and the observations were made by another, confirmed by myself, and demonstrated to many others. It is not, however, a solitary case. I have met with several others; indeed, the day on which I am writing I examined a urine which gave no precipitate with nitric acid, but gave a decided precipitate with heat and acetic acid, with Roberts's brine test, with picric acid solution, with Pavy's pellets, and all Oliver's test-papers. This urine had a sediment of uric acid crystals.

With regard to heat I would point out that it is full of fallacies and failures, especially in connection with the acid and alkali albumens; how liable it is to error I do not think we yet fully know. Some evidence as to this can be obtained from Dr. Kirk's paper already referred to. Heat also fails to detect peptones in the urine, and I shall bring evidence to show that we can no longer afford to overlook them.

Of the new tests the one in which I have found the fewest fallacies is Dr. Roberts's brine test. With this I have been particularly pleased; it is much more convenient to use than nitric acid, it precipitates peptones and some of those forms of albumen to which nitric acid is indifferent, it does not produce a cloud with urates, and I have found it quite as delicate as nitric acid. As far as my experience serves me, it is only liable to the fallacy of producing a precipitate with the gum resins. Why Dr. Roberts should abandon his own test in the way he appears to have done in his recent address to the Glasgow Society, I am unable to conceive; its only evil in his eyes appears to be that it precipitates peptones, and he will have none of these; to my mind this is one of its greatest advantages, but of this more anon.

I have given picric acid a good trial and find it a useful test, but open to some fallacies. It gives a cloud with mucin, and if there is the slightest opalescence before applying the test the urine should be filtered; it gives a cloud with lithates occasionally, which is generally crystalline and easily recognised; there is also the fallacy of the precipitate produced by quinine, which is a serious one as the drug is so often used; it also precipitates the gum resins. Picric acid precipitates peptones

and other forms of albumen which escape detection by nitric acid, and for this I value it.

But I have now met with several cases in which picric acid failed to produce a precipitate in urines in which albumen was undoubtedly present. I may quote another case to which Mr. Harris called my attention; I repeatedly demonstrated the reactions of this urine to students. The urine was obtained on March 19th, 1883, from a patient with acute Bright's disease associated with general eczema. The reaction was alkaline, the sp. gr. 1015, it afforded a considerable cloud with nitric acid by Heller's method and also by heat after the addition of acetic acid; mercuric chloride yielded a precipitate on excess; there was no precipitate with tincture of galls, and no precipitate with picric acid when the urine was acidulated with citric acid or with acetic acid. The reaction continued for some days.

The behaviour of picric acid is often singular; the precipitates obtained by it are quite out of proportion to those obtained by nitric acid; sometimes the picric cloud is much more dense, sometimes much less dense than that produced by nitric acid. This want of quantitative relation requires further investigation, and may prove of importance as indicating varieties of albumen.

There is one great attraction in this test, inasmuch as by the addition of caustic potash it becomes a qualitative and quantitative test for sugar. Mr. Hawkesley has prepared a little pocket case, by the direction of Dr. Johnson, which contains a graduated test-tube, some solid picric acid, and one grain fragments of solid caustic potash by which a rough estimation of the amount of sugar contained in a urine can be made at the bedside. I find the deliquescence of the caustic potash a great practical objection to this, as a pocket test.

Dr. Pavy has quite recently modified and greatly improved his test with potassium ferrocyanide, and citric acid. These, which were formerly combined in one pellet, he has now separated into two pellets; they are issued by Mr. Cooper in a little box adapted to the waistcoat pocket. The citric acid frequently produces a cloud in the urine; this generally indicates the presence of lithates in excess. The cloud is composed of free uric acid; it is produced by the citric acid

pellet alone, before the ferrocyanic pellet is added, and is therefore no longer a source of fallacy. It is readily dissolved by a gentle heat, or by adding warm water. The ferrocyanic pellet is then introduced, and if a cloud is produced it is invariably albumen. This test should only be used in the cold, and if it is necessary to use heat to dissolve the uric acid cloud, it must be employed with great caution, as I find that with a temperature below boiling, some decomposition takes place in the reagents, which then produce a cloud, even in distilled water. Another inconvenience often occurs in the use of this test owing to the effervescence produced by it in some urines. Time must be allowed for this to subside before an opinion can be expressed as to the presence or absence of albumen. *This test does not precipitate peptones and is therefore useful as a check to the two previous tests.* The test is a very delicate one, but I am not quite sure whether it is so sensitive as some of the others.

The little test-papers devised by Dr. Oliver are exceedingly useful and convenient. I have found them also extremely sensitive. The potassio-mercuric iodide I find the most delicate of any; the compound papers of citric acid combined with the albumen precipitant are open to the same objection as Dr. Pavy's compound pellet, namely, that the citric acid causes a cloud with lithates; this paper should therefore always be used separately. Dr. Roberts states that citric acid produces a precipitate with mucin. I have not discovered this, although I have applied the test to several urines cloudy with mucus; it is possible we may both refer to the same precipitate, and this Dr. Pavy attributes to an excess of lithates. The point requires further investigation. With regard to the other test-papers recommended by Dr. Oliver they are all more or less satisfactory. I find none quite so good as the potassio-mercuric iodide; next to that, in my opinion, comes the ferrocyanide; then perhaps the tungstate of soda, which is certainly not nearly so delicate as the others; and last of all the picric acid. The objection to the latter is the small amount of picric acid available, as the reagent requires to be in excess; moreover, it takes some little time dissolving out of the papers.

As a matter of convenience for bedside observations I have had a small pocket case prepared by Messrs. Down Bros., in

which I have arranged Dr. Pavy's pellets for albumen and sugar, one or more of Dr. Oliver's paper-tests, selecting the potassio-mercuric iodide with separate citric acid papers as the best albumen precipitant, and the indigo-carmin papers for the detection of sugar, a pipette, two test-tubes, a taper, matches, litmus papers, and two specific gravity balls to detect a specific gravity above 1025 or below 1010. I find this practically a convenient combination and sufficient for all purposes. The tests are all dry, and are selected to check one another; thus the picric acid and potassio-mercuric iodide papers throw down peptones, paraglobulin, and quinine, while the ferrocyanic pellets give no reaction with these substances.

PART IV.—THE CLINICAL SIGNIFICANCE OF ALBUMINURIA.

Having considered the pathology of albuminuria and the methods of detecting it, we have now to deal with the question of its clinical significance. Concerning this there can be no doubt that our opinions have undergone a great change within the last few years. For my own part, I am quite convinced that we may have albuminuria with practically healthy kidneys. The frequency of its occurrence in boys and young men having been pointed out by Sir William Gull in the course of a discussion at the Royal Medical and Chirurgical Society in 1878, attention was first seriously drawn to the matter by Dr. Moxon's article in the 'Guy's Hospital Reports' of 1878, and since that date contributions to the subject have been very numerous. Leube having been struck by its frequent occurrence in patients who had no symptom of renal disease, made observations on 119 soldiers and found it present in 19 of these, or 16 per cent.; an account of his observations appears in 'Virchow's Archives' for 1878. In the following year Dr. Saundby published some remarkable observations in the 'British Medical Journal,' in which he stated that having examined 145 male patients who attended the out-patient room *seriatim*, he found albumen in the urine of 105 of this number, or 72·4 per cent. This experience has been considered quite exceptional by subsequent writers, and although I am very familiar with Dr. Saundby's exceed-

ingly careful and reliable work, I have always found some difficulty in accepting these observations. It is possible that acetic acid may have produced some fallacy with mucin; it would be of great service if Dr. Saundby, or some equally reliable observer, would repeat these observations, using either heat and nitric acid as Leube did, or else Roberts's brine test, which I believe to be more delicate and trustworthy.

Examples of this temporary and intermittent albuminuria without other evidence of renal disease have been recorded by Clement Dukes, Rooke, Edlefsen, Fürbringer, Griffiths, Johnson, and Kinnicutt. I would point out that while all of these regard the condition as a not unfrequent one, yet they by no means go so far as to say that it is a normal one, or that it occurs in a majority of the urines examined. It is true that Dr. Roberts and others have asserted that some microscopical traces of albumen can be demonstrated in every specimen of normal urine, yet that is not the condition we are now dealing with; we have to consider cases in which albumen is found to a well-marked extent in the urine by the use of the ordinary clinical tests, and its appearance there is sufficiently rare and remarkable to make it considered noteworthy by experienced clinical observers. It is also desirable to remember that these results were obtained with the old-fashioned tests, heat and nitric acid, they were not albuminurias discovered by the new and more delicate tests, they cannot be classed as mere "peptonurias," a condition which it is becoming fashionable to speak of and to dismiss as of no importance; in all of these cases the urine contained what Dr. Roberts calls "morbid albumen." But the mention of this distinction leads me to pause for one moment to consider the clinical significance of those reactions which are commonly ascribed to peptones in the urine. It has been asserted that we only require to recognise in the urine the presence of serum-albumen, and globulin, and that for all practical purposes the existence of any other proteids in the urine may be ignored. I have already pointed out that we do not know what forms of albumen are found in the urine, and that true serum-albumen probably undergoes remarkable changes in that fluid. I have frequently obtained evidence of the presence of albuminous bodies in the urine by means of picric acid and

the brine test when heat and nitric acid have failed to afford any indication, although the presence of albumen was either previously or subsequently demonstrated by these latter tests also. In these cases there was every reason to suspect morbid changes in the kidneys, but the indications afforded by the urines on these occasions would have been overlooked if heat and nitric acid had been relied upon as tests for albumen. This form of albumen, which escapes the ordinary tests and is detected by picric acid and the brine test, I have found to be particularly liable to occur in cases of acute renal disease following scarlatina and other similar conditions. Almost any day I can find such reactions in urines of scarlatinal patients at the Fever Hospital, who either have had, or who subsequently develop, ordinary albuminuria.

I shall have to refer later to other cases of chronic Bright's disease which gave no evidence with the heat and nitric acid tests, but afforded indications with the more delicate reagents.

From these and similar observations I am led to conclude that peptones or other easily diffusible forms of albumen in the urine (which escape the ordinary tests with heat and nitric acid but are detected by other reagents) afford indications we should not neglect, for they appear to be often of equal importance with the albumens detected by the old methods. Either we can afford to disregard albuminuria altogether, because it is sometimes found with healthy kidneys, or else it behoves us to use the tests which are most likely to detect any form of albumen that may appear in the urine.

I believe that the appearance of any form of albumen in the urine indicates undue stress upon the renal circulation. It means in fact increased pressure and distension of the glomerules or slowing of the circulation within them. The only exception I would make to this rule is that possibly an abnormal albumen, such as peptones due to imperfect digestion, may be occasionally present in the blood and appear in the urine. It appears probable that there are forms of albumen constantly present in the blood that are both diffused and filtered more readily than ordinary serum-albumen, immature albumens perhaps, and that on the first appearance of delay in the glomerular circulation these may appear in the urine, that if the condition persists and increases, ordinary

serum-albumen is very likely to follow. In connection with this subject it is well to remember that Gamgee states, on the authority of Drosdoff, as the result of the most recent research upon the matter, that peptones cannot be detected in healthy blood. On the other hand, paraglobulin is always present in the blood, it is an albuminous body much more readily diffused or filtered through an animal membrane than ordinary blood serum, it is readily converted into an acid or alkali albumen and therefore might easily escape detection by heat, by which it is otherwise precipitated, it is precipitated by sodium chloride in excess and this reaction may account for the cloud produced by the brine test in urines in which heat and nitric acid fail to give a precipitate. There would therefore seem to be good ground for the belief that the term "paraglobinuria" might be substituted for "peptonuria," though for my part I prefer to describe all alike as albuminuria.

This disturbance of the glomerular circulation may be a very slight thing, a purely functional condition, or it may be a very grave thing if it depends on organic changes. That we may form some idea of the occurrence of albuminuria in so-called healthy persons I take for consideration my own experience, during the last year, of the examination of practically healthy persons. I have notes of the examination of seventy-seven persons, all males, who came before me during twelve months for examination previous to life-insurance, or for similar purposes. In all of these cases I carefully examined the urine and I found albumen, in some form or other, present in twelve cases, that is in 15.5 per cent., almost exactly the same result as Leube obtained; he discovered albuminuria in 16 per cent. of 119 soldiers, but he had the advantage of examining the urine many times, and on various occasions. I may mention that all my examinations were made in the forenoon, at the time, therefore, when we are most likely to detect albuminuria, if it is liable to occur. It should be noted, however, that five of my cases gave no precipitate with heat or nitric acid.

CASE 1.—A. R. D—, æt. 26, examined April 7th, 1883. A man stated to be perfectly healthy. There is no gout in his family. Ill nourished, narrow chested, strumous looking. On

March 23rd he had travelled from Folkestone to Boulogne, and had been on the deck of a steamer for three or four hours. It was a bitterly cold night. His urine was found to contain a cloud of albumen by nitric acid and heat. His sphygmographic tracing (Fig. 2) showed contracted vessels, but no

FIG. 2.



Pressure 2 oz.

marked increase of pressure. No oedema, or other signs of Bright's disease. I advised him to consult his doctor, and to return again for examination in one month.

This person did not call again for further examination until March 27th, 1884; it was an exceedingly cold day, with an east wind. He said that he had had good health ever since he was here, a year ago, and that he had not required any medical advice. He looks, perhaps, a little puffy below the eyes, but this is only noticeable on critical examination. His sight is good, except for a little myopia. His pulse is non-persistent, but small, the vessels being contracted. *Urine.*—Strongly acid, of natural colour. A considerable precipitate both with picric acid and the brine test, which largely redissolves with heat. A similar precipitate with potassio-mercuric iodide and citric acid paper. No precipitate with heat, nitric acid, or with Pavy's citric and ferrocyanic pellets. With nitric acid the urine gives a dark red ring of colour, and with chlorate of potash and hydrochloric acid a well-marked indican reaction is obtained.

I should prefer to see this person again before giving an opinion about him. I cannot but think, however, that his kidneys are *tender*, though there probably is not at present any definite organic disease.

CASE 2.—W. H. N—, æt. 44, examined April 25th, 1883. A traveller in the wine and spirit trade. Insured seven years ago as a first-class life. Drinks both beer and spirits freely. A fine, tall, but somewhat full-blooded man. Height 6 ft. 2½ in. Weight 16 st. 4 lbs. He has had one attack of gout, nine months ago. Liver enlarged, dulness extends one and a

half inches below ribs. Urine highly acid, contains a decided cloud of albumen.

I saw him again on May 4th, that is, nine days afterwards. He looked clearer and brighter; had not taken any beer; had been staying in Devonshire and drinking cider. *Urine*.—Acid, sp. gr. 1028. Faint cloud of albumen with brine test, none with nitric acid even after standing; a scarcely appreciable cloud with heat. Faint cloud with picric acid; a faint cloud with ferrocyanide and citric acid. Unfortunately I did not take a tracing of his pulse. It did not feel long or hard. Advised to postpone and watch.¹

CASE 8.—N. R. E—, æt. 56, a medical practitioner, examined May 26th, 1883. General health good, but liable to bronchitis occasionally. Very good family history. He had whooping-cough for second time three years ago. Has lately been depressed and overdone about money matters. Can run well and plays lawn tennis. He looks a little pale. Has a slight hæmic bruit over base of heart. Impulse in normal position. Pulse soft, no sign of increased arterial pressure. Suffers from dyspepsia (flatus and gastralgia). *Urine*.—Acid, sp. gr. 1018. No precipitate by nitric acid (Heller's method) or by heat; faint precipitate with brine test and picric acid. It gives a play of colours with nitric acid, probably due to indican. Seen again on June 25th, a month later. Urine gives the same reactions as before. Taken as a fair average life.

CASE 4.—S. M. J—, æt. 34, also a medical practitioner, examined June 21st, 1883. His general health has been perfect. Never a day's illness except scarlatina when aged twelve, and croup when a child. Heart and lungs normal.

FIG. 8.



Pressure 1 oz.

No sign of high arterial pressure (Fig. 8). *Urine*.—Acid, too

¹ Since writing this paper I have heard that this patient has had another attack of gout and is now severely ill. His life is now quite uninsurable.

small a quantity to obtain the specific gravity. No cloud with nitric acid. A precipitate with heat, which clears almost completely with acetic acid. It gives a decided precipitate with brine test, and a very slight precipitate with picric acid. Taken as a fair average life.

CASE 5.—H. M. O'D—, æt. 28, a hair-dresser, examined November 15th, 1883. Much too stout for his age. Looked as if he drank too much beer, and admitted that he does do so. His pulse was 124, small, and soft under examination, while a loud systolic bruit was heard at the lower part of sternum. It was not heard at upper part of sternum or only faintly there. It faded over pulmonary area and at apex. It was not heard in trachea or in large vessels of neck. Apex beat imperceptible. There was no undue impulse or increased cardiac dulness, no cyanosis or dyspnoea. He is a good swimmer, and can keep under water half a minute. I thought it might be temporary tricuspid regurgitation. *Urine.*—Contained no sugar. No precipitate with heat or nitric acid. A slight precipitate with brine test, and with potassium ferrocyanide, and citric acid. None with picric acid. Refused on account of alcoholism combined with fat and other symptoms.

CASE 6.—H. D—, æt. 25, a clerk, examined November 28th, 1883. A healthy-looking man. Good runner, fond of athletics. His father died, aged fifty-five, from collapse after severe vomiting and diarrhoea. He is liable to attacks of severe headache and vomiting. For these he consulted the late Dr. Hilton Fagge in January last, who then examined his urine and stated that it was healthy. In May last he had an attack of severe constipation, with colic and abdominal pain, and vomiting; this lasted for three days. He has had no recurrence of it. He has always been very liable to constipation both before and after this. *Urine.*—A slight precipitate with heat, nitric acid, brine test, and picric acid. Action of the heart powerful. Apex not displaced; sounds normal. Sphygmographic tracing of pulse (Fig. 4) does not show markedly high pressure, though some tendency in this direction; it requires four ounces to develop it. He came

again on January 10th (about six weeks after). He stated

FIG. 4.



Pressure 4 oz.

that he had called on his doctor, as I directed him, the day after his last visit, that the doctor had examined his urine carefully, and could detect no trace of albumen. This day I found no precipitate with nitric acid, but it gave a purple discolouration. There was a precipitate with heat which completely cleared with one drop of acetic acid. There was a very faint cloud with the brine test and picric acid. January 21st.—He had had a bilious attack the day previous, feeling sick with headache in the morning, retching all the afternoon. Vomiting of bile at 6 p.m., and then improvement. This day he feels well. No headache. *Urine*.—Cloud with nitric acid and with heat, not clearing with acetic acid; precipitate with the brine test. Dr. Calthrop, of Highbury, who has attended him since a child, kindly wrote to tell me that he had tested the urine after the next bilious attack nine days later, and had obtained no precipitate with either heat, nitric acid, or picric acid; the sp. gr. on this occasion was 1025. Seen again on March 4th his urine then gave a precipitate with nitric acid and brine tests, which only appeared after it had been allowed to stand for an hour.

In this case I believe the albuminuria is dependent on the nerve storms. I have seen others which indicate that it is probably a not unfrequent accompaniment of such attacks, although not often searched for. There is no evidence to show that the lives of patients who are subject to such attacks are shortened to any appreciable extent, in fact, I know of several persons now advanced in years who have been very liable to them. I recommended that the addition of seven years would be sufficient to cover the uncertain risk in this case.

CASE 7.—J. B—, æt. 35, a commercial traveller in the

whisky trade. Examined December 14th, 1883. Fond of social evenings, which are often followed by "bilious attacks." A tall, spare, healthy-looking man. He is liable to boils. All his organs were found to be normal, as far as could be ascertained by physical examination. He is just recovering from a severe bilious attack. *Urine*.—A precipitate with heat, nitric acid, and brine test. Reaction acid. Advised to postpone and watch.

CASE 8.—L. D. G—, æt. 47. Examined January 25th, 1884. A stout, heavy man, now an abstainer. Chest expanded imperfectly on account of his being overloaded with fat, but his organs appeared healthy and performed their functions well. *Urine*.—No precipitate with nitric acid, but a very slight cloud with the brine test. Recommended to accept without addition.

CASE 9.—L. McK—, æt. 22. Examined January 28th, 1884, for a clerkship. Has always had good health, no minor ailment of any kind; his last serious illness was scarlatina ten years ago, which left no sequelæ. He is skilled in throwing the hammer and putting the stone, and has taken some prizes. Lungs and heart normal, apex beat normal. Pulse 64, good volume, soft. *Urine*.—No precipitate with heat or with nitric acid. A marked precipitate with brine test and a faint one with picric acid; the former does not clear with heat; the precipitate with picric acid perhaps clears a little but not completely, I doubt if it does more than diffuse. I gave a certificate of good health.

CASE 10.—B. S. J—, æt. 30, a schoolmaster. Examined February 27th, 1884. A remarkably healthy-looking, spare muscular man, in first-rate condition and training. Very athletic, has taken many prizes for bicycling. Has never had any illness. Father, mother, and seven brothers and sisters all living and healthy; has lost none except in early infancy. *Urine*.—Strongly acid. No cloud with nitric acid, but a purple discolouration. A faint but distinct cloud with heat, not diminished by acetic acid. A faint cloud with picric acid and with brine test. A remarkably good cloud with

ferrocyanic pellets. No increase of arterial pressure. Recommended to accept without addition.

CASE 11.—B. H. W—, æt. 23. Examined March 8th, 1884. A very nervous, emotional man. Pulse 100, soft. Slight excitement bruit with first sound of heart, best heard at lower part of sternum and internal to apex. Impulse in normal position. *Urine*.—Alkaline. Dense precipitate with heat, cleared completely by acetic acid. No precipitate with nitric acid. Faint precipitate with brine test and with ferrocyanic pellets. None with picric acid. Recommended to accept without addition.

CASE 12.—T. L—, æt. 41, an engraver. Examined March 31st, 1884. A neurotic and dyspeptic man; very thin, spare, and ill-conditioned. He suffers much from flatulent distension. His father died of phthisis. *Urine* gives a faint cloud with brine, picric acid, ferrocyanic pellets, heat and nitric acid; the last gives a purple discolouration, and the cloud is very faint, it only appears slowly, some while after addition of the acid. Heart sounds normal, except that the aortic second sound is accentuated. Impulse in normal position. His pulse is difficult to appreciate correctly by the finger; it appeared rather long. On applying the sphygmograph the following tracing (Fig. 5) was obtained; it shows a

FIG. 5.



Pressure 1 oz.

very decided increase of arterial pressure, the tidal wave is much prolonged. The best tracing was obtained with one ounce of pressure; it required nine ounces to compress it. Recommended to refuse or only to accept with a large additional premium.

In all of these cases, except Cases 4, 9, and 10, there is

very little difficulty in tracing a cause for the albuminuria, although every one of these persons considered himself in perfectly good health. It is not surprising that Leube found albuminuria in 19 out of his 119 soldiers; probably similar slight causes might have been traced in his cases if they had been carefully sought for. It may be remarked that although I did not usually consider the risk increased in the case of those whose urines gave reaction with the more delicate tests, but none with heat or nitric acid, yet it is noticeable in three cases, Nos. 1, 2, and 6, that precipitates with heat and nitric acid appeared to alternate with precipitates obtained by the more delicate tests.

We have next to consider the significance of albumen in the urine under the various circumstances in which it may be found. Albuminuria may be divided into several groups, and our first endeavour should be to determine to which of these groups the case under consideration belongs.

I.—Cases of fully developed Bright's disease with albuminuria, dropsy, cardio-vascular symptoms, and perhaps retinal changes, anæmia, dyspepsia, &c. In such cases as these our prognosis must always be a grave one, though I have known some remarkable instances of recovery from apparently the most desperate conditions, and of lives prolonged for many years although the symptoms of Bright's disease were unmistakable.

There are many cases in which we have every reason to suspect the organic changes of Bright's disease, but in which our prognosis should be most guarded. Such cases with care will continue in very tolerable health, although they may not be able to follow their usual occupations as actively as others. In some albuminuria will exist for very long periods, even in large amount. In my opinion we are justified in giving a fairly hopeful, though properly guarded, prognosis in all cases of Bright's disease, except those in which albuminuria, high arterial pressure, dropsy, and progressive cachexia have all been present for three months, or in which there is evidence of grave cardiac incompetency. We cannot of course exclude the risk of accident arising from such temporary conditions as suppression of urine, oedema of the lungs, and the various symptoms usually ascribed to uræmia.

CASE 13.—I am acquainted with a gentleman, now living and aged 72, in whose urine Dr. Sibson discovered albumen twenty-seven years ago, the patient then being 45 years of age; it is possible that it may have been present for some time before that; it has been constantly present in considerable quantities ever since. As the condition was associated with gout, and there were no other symptoms of Bright's disease, Dr. Sibson gave a favorable prognosis, which has been more fully verified than he could at the time have expected. I have no evidence as to the condition of the arterial pressure in this case. There has never been any dropsy or other grave symptom; the gentleman has lived an active life, and except for occasional attacks of gout, has enjoyed good health. Some four years ago he suffered from symptoms which closely resembled angina pectoris, but these have diminished in severity and his general condition is fairly good for his age.¹

CASE 14.—Another case which I have still under observation, is that of a woman over fifty years of age; three years ago she was suffering from typical, though slight, renal dropsy, with albuminuria, very high arterial pressure, and, as I believe, some hypertrophy of her heart. This person is now apparently enjoying good health and is actively occupied, but I have no doubt that she has chronic Bright's disease, about which I thought more gravely three years ago, when I discovered it, than I do at present. The albuminuria is not constantly present in this case, and I have had no opportunity of examining the urine for a long time past.

II. *Cases in which the cardio-vascular symptoms and changes of Bright's disease exist with little or no albuminuria.*—I have described in two previous papers in these 'Reports'² this very large and important group. The clinical aspects which such cases may assume are very varied, and they need not be dwelt upon now. As examples of these cases I may refer to those from which the next two tracings were obtained.

¹ Since writing the above the gentleman has died, somewhat suddenly, from pneumonia, combined with cardiac failure.

² 'Guy's Hospital Reports,' 1879 and 1882.

CASE 15.—Fig. 6 was obtained from a rather stout man, aged about 50, with dyspepsia and a gouty history, who had been subject to phthisis many years ago, but had recovered. His urine gave a cloud with the brine test, but none with heat or nitric acid ; his pulse tracing exhibited a very decided

FIG. 6.



Pressure 8 oz.

increase of arterial pressure, and I think it probable that his kidneys are becoming granular. The indication afforded by his urine would have been overlooked if the ordinary tests had been relied upon.

CASE 16.—Fig. 7 is the pulse tracing of a woman under the care of Dr. Brailey for neuro-retinitis ; she is puffy-looking, and her breath is short. Her urine on one occasion gave a cloud with brine test and picric acid, but none with heat and nitric acid ; on two other occasions it gave no abnormal

FIG. 7.



Pressure 4 oz.

reactions. Her pulse tracing shows a very considerable increase of arterial pressure. I conclude from these symptoms, together with her retinitis, that she probably has granular kidneys. Here again the urine afforded no indication with the ordinary tests.

Our prognosis in such cases as these need not, I think, be grave ; the symptoms warn us of possible dangers, but they may be years distant ; the chief advantage of recognising the condition is the key it gives us to the general management

and treatment of the case, and of the most effective way to deal with the little ailments that may from time to time arise.

The next case is perhaps out of place in this paper, no albumen having been found in the urine; I venture to introduce it on account of its great value as a type of this class, and as an illustration of the common sequence of pathological changes in these cases.

CASE 17.—In July, 1874, that is, ten years ago, I first became acquainted with Dr. Z—, from whom the tracing reproduced in Fig. 8 was obtained. He was at that time

FIG. 8.

Pressure 5 oz.

suffering much from neuralgia and other symptoms which were thought to be indicative of latent gout; he was liable to anginal attacks and to dyspnoea on active exertion. His tracing at the time showed exceedingly high arterial pressure, and his health was a source of grave anxiety to his friends. I regarded him as suffering from gouty kidney and its attendant cardio-vascular changes, and I thought that the condition of his circulation indicated probable failure through his heart. By means of care, rest, and good management he has lived fairly comfortably for the last ten years, and it is hoped that he may continue to do so for some time longer. On October 5th, 1888, I received a letter from Dr. Shingleton Smith, of Bristol, under whose care Dr. Z— has been for some years, in which he says, "Some months ago you mentioned to me the fact that at one time our mutual friend Dr. Z— had a hard pulse, indicative of high tension, as shown by the blunt square-topped sphygmogram. I took a tracing of his pulse to-day and it occurred to me that you would be interested in comparing its present with its former appearance." In another letter about this patient Dr. Shingleton Smith says, "It is very singular that one never finds even a trace of

albumen in the urine. I have several times examined it, but always with a similar result."

In reply to some further inquiries, which I recently made for the purpose of introducing the case into this paper, Dr. Shingleton Smith sends me the following valuable letter, dated March 31st, 1884:—"I have seen Dr. Z— to-day, and taken the enclosed tracings (fig. 9) which do not materially differ from the one which I took some months ago. His age is now seventy-one. He has never had a definite attack of acute gout, but has looked upon various indefinite pains and slight nodosities of fingers as gouty for the last fifteen years. He has constantly dosed himself with potash ever since, whenever the urine became turbid with lithates. He has had frequent attacks of prurigo, but worse than ever during the last six months. The skin has sometimes been so inflamed and swollen as to give slight pitting on pressure, but there has never been any true œdema from heart or kidney. The urine to-day is clear, sp. gr. 1020, acid, no trace of precipitation with heat, nitric acid, picric acid, or brine solution; no sugar. The heart is now fairly regular and strong, but a rough systolic murmur is audible at base, another at apex of a softer character, and at xiphoid cartilage a short, faint, soft, diastolic murmur is heard immediately following a very loud accentuated second sound. The apex is just below the nipple, scarcely out of the normal position. Lungs and liver normal. The optic discs are healthy, and I can see no indications of any present or past retinal hæmorrhages. The attack of left hemiplegia of eighteen months ago has left very slight impairment of articulation, but no other defect. His general health is fairly good, he walks out alone every day, and is mentally as good as ever. You know that he used to have attacks of cardiac angina, and also biliary colic, but neither for the last two years. I look upon him as an epitome of pathology, but cannot help thinking that his kidneys are better than most of his organs.

"April 1st, 1884.—P.S. I have again examined the urine by daylight, and do not get even the faintest cloudiness with picric acid or with Roberts's brine. I do not happen to have any of Oliver's papers, as I have been content to trust to picric acid or to the salt solution. The urine whilst cold is just

a little cloudy, but clears completely with heat; the cloudiness is due to fine crystals of urates, which are visible under the microscope, but no granular casts could be seen. In order to be sure of the absence of albumen, I had to warm the urine before applying the albumen tests, but having done so all the tests agree that there is no trace."

The pathology of this case is to my mind perfectly clear, and it is typical of the sequence of events that I have watched in many cases. It is a case of Bright's disease in which the cardio-vascular symptoms are prominent, while the renal are in abeyance. It is probable that the kidneys are more or less red, granular, and contracted, but it is quite possible that they may be but little changed. It is certain that his arteries are universally thickened and the heart hypertrophied from the prolonged existence of high arterial pressure. There is probably some shrinking of the aortic valves from chronic atheromatous changes, which may have crept on to them from the aorta; the mitral valve is probably thickened, and also its *chordæ*. In fact, as I anticipated ten years ago, the strain on the circulation has injured the heart, but the changes have been remarkably slow and gradual, owing to the care and discretion he has shown in the management of his health; if Dr. Z— had continued in full work I am convinced that he could not have lived nearly so long. He has probably added ten years to his life by his wise discretion, and may possibly add a good deal more. The case is most valuable as showing the practical arrest of advancing disease by the adaptation of life to the requirements of health.

The tracing reproduced in Fig. 9 was obtained by Dr.

FIG. 9.

Pressure 3 oz. Taken by Dr. Shingleton Smith with Dodgeon's sphygmograph.

Shingleton Smith on the occasion of the visit, the results of which are detailed in his last letter; it is practically the same

as the tracing obtained in October last. Though this tracing was obtained by means of Dr. Dudgeon's sphygmograph and is not therefore strictly comparable with my own, yet the general outlines are comparable. The great difference between the pulse now and ten years ago lies in the reduction of the tidal wave; this was previously long and sustained, and afforded evidence of high arterial pressure and a labouring, powerful heart. Since then aortic regurgitation has taken place, the overfull arteries have proved too great a strain for the aortic valves, which have given way under it and allow a constant reflux. I suspect that the mitral valve also allows some escape backwards, and that the pulmonary system and the veins have accommodated themselves to some engorgement; in fact, that the increased pressure, previously confined to the arterial system, is now shared more equally by arteries, pulmonary system, and veins. The arterial pressure being thus reduced, the tidal wave is shortened and the ventricular effort diminished. I do not think that this will prove a permanent advantage, although it may be a temporary relief; it probably does not affect the left ventricle so seriously as primary aortic regurgitation does a normal heart, because in this case the ventricle was previously hypertrophied and partly prepared to resist the tendency to dilatation produced by aortic regurgitation.

III. *Cases of nephritis in which the albuminuria is severe, prolonged, and accompanied by dropsy, but in which there is no increase of arterial pressure.*—The most important point concerning all cases of albuminuria is to ascertain whether or not the arterial pressure is increased. Accurate information on this point is essential to a just prognosis. For example, the

FIG. 10.



Pressure 2 oz.

case from which Fig. 10 was obtained was that of a man

admitted into Guy's Hospital with a sharp attack of acute Bright's disease; he had much albumen in his urine, and some pyrexia; his pulse was never one of high pressure, and it remained soft during his recovery. I regarded it as a purely renal affection, due to chill and not to a blood disorder. He made a good recovery without giving cause for any anxiety. I might quote many similar cases.

CASE 19.—Again the case from which the tracing reproduced in Fig. 11 was obtained was that of a gentleman who had severe

FIG. 11.

Pressure 4 oz.

and prolonged albuminuria after scarlatina, from which he suffered in 1876. He was then thirty-eight years of age; the albuminuria existed off and on for four years, in spite of most careful treatment by diet and other means. The albumen disappeared on several occasions while residing in Egypt and Algiers during the winter and as frequently reappeared. At times it was associated with cedema and other grave symptoms. His pulse, of which I obtained tracings on several occasions, never afforded evidence of high arterial pressure; it was uniformly large, soft, and non-persistent. The tracing in Fig. 11 was obtained in August, 1878, when his urine was highly albuminous; it is that of a perfectly healthy pulse; his heart also afforded no evidence of increased arterial pressure. On the absence of the cardio-vascular signs of Bright's disease, I founded an opinion that the disease of his kidneys was a purely local affection, and that he was not suffering from the constitutional condition which is the essential part of Bright's disease. I thought it probable that the albuminuria might be caused by a local patch of disease in one kidney, which had probably been more seriously affected than the rest of the organ, and had recovered less completely; I have seen such

conditions in the post-mortem room, and believe that they occasionally account for some cases of prolonged albuminuria, without the other symptoms of kidney disease. The subsequent occurrence of slight dropsy in this case seems to indicate, however, that the kidneys were more generally affected. I constantly maintained a good prognosis throughout the case, and it proved to be correct. The albuminuria disappeared in 1880, and has never since returned; the patient remains free also from any of the cardio-vascular signs or changes of Bright's disease.

CASE 20.—Another similar case was that from which the tracing reproduced in Fig. 12 was obtained. It is the tracing

FIG. 12.



Pressure 3 oz.

of the pulse of a gentleman aged about twenty-two at the time at which it was taken, now ten years ago; he had suffered from acute nephritis for fifteen weeks. His urine had been highly albuminous and bloody, at one time almost suppressed; he had general dropsy, double hydrothorax, fluid in the peritoneal cavity, and only narrowly escaped with his life. I do not know what had been the condition of his pulse during the earlier stages; when this tracing was obtained he was recovering, though his urine was still albuminous and the dropsy had not disappeared. His pulse tracing is one of low arterial pressure, quite unlike that of Bright's disease in its more severe forms, and for this reason I formed the opinion that the case would not go on to a chronic form of Bright's disease but would make a good recovery. This prognosis proved correct for the gentleman made an excellent recovery and has ever since been in the active practice of his profession. From these and similar cases it appears to me that we have a most valuable and important guide to prognosis afforded us. I do not assert that the pulse tracing can be relied upon alone; there are cases of Bright's disease

which die through the failure of the kidneys though the arterial pressure is low, and some with really high arterial pressure occasionally afford a *tracing* of apparently low pressure. This form of tracing may be produced in Bright's disease by dilatation of the heart and secondary incompetency of the mitral valve; the regurgitation shortens the systolic expansion and does away with the prolonged tidal wave which is so characteristic of the pulse of high arterial pressure; to the finger such a pulse will be *persistent*, the artery tense and cord-like. With an intelligent appreciation of the other facts of the case, I believe that we may safely look to the sphygmograph to give us most valuable indications in many cases of Bright's disease, information that we can obtain in no other way, and which must always be of the utmost importance to our patients.

IV. *Cases of slight albuminuria in which the arterial pressure is increased.*—An excellent example of this class is afforded by the following case.

CASE 21.—B. G. H—, a boy, æt. 12, was brought to me for a certificate of health previous to admission to a public school in April, 1882. He is a slight, rather delicate-looking boy, but had enjoyed very good health previously and was considered in his usual health at the time of my examination. I could detect no evidence of any kind of disease about him, except a very well-marked cloud of albumen in his urine, which was obtained by the ordinary tests, heat and nitric acid. In the absence of other symptoms I regarded the albuminuria as having but little importance and gave him the required certificate of health. I did not obtain a tracing of his pulse on this occasion. In April, 1884, that is, two years afterwards, I again had an opportunity of examining this lad; he had enjoyed perfectly good health while at school and looked stronger and better than he had two years previously. To my surprise I found that his urine was still albuminous, if anything it contained rather more than before. In consequence of this I took a tracing of his pulse, which is reproduced in Fig. 13; it shows a pulse of decidedly high arterial pressure, and I cannot but fear that the condition of his pulse, taken together with his albuminuria, threatens the

development of more serious symptoms on very slight provocation. I believe that Bright's disease is an ever-constant

FIG. 13.



Pressure 3 oz.

danger in such a case, though it is likely enough that there are no organic changes at present. It is possible that the condition may change as life advances and that he may grow out of this, as young people grow out of many constitutional conditions predisposing to sundry small ailments such as tonsillitis, bronchial and gastro-intestinal catarrhs, boils, &c. Meantime I contented myself with advising that he should constantly wear flannel next his skin, be careful to avoid chills and exposure, and above all things keep his bowels regular and well open. Another excellent example of this class is afforded by Case 12 already quoted.

V. Cases in which there is slight or intermitting albuminuria, but no other symptom of Bright's disease. The arterial pressure not being increased.—Cases 1 and 6 may be taken as examples of these; I have seen other more marked ones. In such cases I should be inclined to regard the kidneys as tender, and the patient likely to develop the renal side of Bright's disease; I should be especially anxious if the patient developed these symptoms when over twenty-five years of age. In a case of intermittent albuminuria, long persistent, in which circumstances favoured the arrangement, I should advise a sea voyage, and if the symptom still continued permanent residence in a warm climate. In such a case one would regard acute nephritis as an ever-impending danger.

Lastly, we must remember that various conditions may be recognised and accepted as sufficient causes for albuminuria, such as debility, anæmia, heart disease, disordered digestion, and some neurotic disturbances. All of the conditions enumerated may give rise to what I believe to be a purely functional albuminuria, the kidneys being practically healthy.

The symptoms which accompany the albuminuria, taken together with the absence of other symptoms of Bright's disease, sufficiently indicate its nature. Several examples of this class occur among my first twelve cases.

In all cases of albuminuria of a doubtful nature it is necessary to carefully watch the condition of the urine. When doing so it is well to remember, first, that albuminuria can be produced as a temporary condition in healthy persons; second, that its presence in Bright's disease is variable and its quantity still more so. It has been ascertained that albuminuria can be produced in healthy persons as a temporary condition by cold bathing,¹ by a diet consisting largely of egg albumen,² and by excessive muscular exertion;³ all these causes will also increase the amount of albumen in cases of Bright's disease. Other conditions which cause a variation in the amount of albumen present in the urine in Bright's disease have been carefully investigated by several observers, and especially by Dr. Saundby,⁴ whose interesting article contains an excellent summary of these conditions. The quantity of albumen is increased by a highly nitrogeaneous diet,⁵ by rising from bed,⁶ and it is always present in larger quantity in the urine passed in the forenoon, after breakfast, than at any other period of the day.⁷ These circumstances should always be borne in mind, and if we wish to put the kidneys to the test, we should not be contented to examine the urine passed immediately on rising in the morning, but rather select a specimen passed in the forenoon, after a moderate amount of exercise has been taken.

Finally, I would again express my firm belief that no trustworthy opinion can be formed concerning any case of albuminuria without the use of the sphygmograph. Notwith-

¹ Dr. George Johnson, 'Trans. of Clin. Soc.,' vol. vii, 1874, and by myself, 'Practitioner,' July, 1875.

² Many authors, vide *supra*.

³ Leube, 'Virch. Archiv,' 1878, lxxii.

⁴ 'British Medical Journal,' June 5, 1880.

⁵ Parkes, 'Med. Times and Gaz.,' 1852 and 1854; Pavy, "Gulstonian Lectures," 'Lancet,' 1863; Drs. Sparks and Mitchell-Bruce, 'Trans. Roy. Medico-Chirurg. Soc.,' vol. lxii.

⁶ Saundby, loc. cit., and myself, 'Trans. Roy. Medico-Chirurg. Soc.,' 1874.

Moxon, 'Guy's Hospital Reports,' vol. xxiii, 1878; Saundby, loc. cit.

standing a careful examination of the pulse by the finger I have been repeatedly deceived as to its significance. It is in such cases as these that the sphygmograph is most useful; it is indeed indispensable to their correct appreciation. I do not mean to affirm that every case of albuminuria in which the arterial pressure is found to be high is necessarily a case of Bright's disease; on the contrary, I know that such a condition is not unfrequently a temporary one, and can be caused by neurotic disturbances; but I submit that the co-existence of these two symptoms is a matter of the utmost importance. If either of them be present for a considerable period it may be taken as an indication forewarning Bright's disease, but if they should co-exist persistently the case can only be regarded as one that may be incipient, but is probably advancing organic disease.

ON TRANSFUSION OF BLOOD

FOR

PUERPERAL HÆMORRHAGE.

BY ALFRED LEWIS GALABIN, M.D.

THE records of the Guy's Hospital Lying-in Charity have not hitherto shown very encouraging results with regard to the saving of life in puerperal hæmorrhage by the transfusion either of blood or any other liquid. Death from hæmorrhage has, indeed, not been very unfrequent, notwithstanding the improvement which has been generally introduced into obstetric practice of late years as regards the management of the delivery of the placenta by external pressure, and the avoidance of any traction whatever upon the funis. Out of the cases recorded in the last report for twelve years,¹ comprising 23,498 deliveries, there were twenty-one deaths from hæmorrhage. Of these, eleven were from post-partum hæmorrhage, four were cases of placenta prævia, five of "accidental" ante-partum hæmorrhage, and one of inversion of the uterus. Besides these there were four cases of rupture of the uterus or vagina, in which death quickly followed from hæmorrhage combined with shock.

During this period transfusion was performed in only five cases, and life was not saved in any one. It was sometimes found impossible to obtain a donor of blood, and on this account, in two instances, a saline solution was used. A tem-

¹ 'Guy's Hosp. Rep.,' 1876, vol. xxi.

porary benefit was observed from this, but the patients ultimately died. In one case transfusion was performed by Dr. Braxton Hicks with his own apparatus, the blood being kept from clotting by the addition of a solution of phosphate of soda, but the patient did not recover. In a second case immediate transfusion was performed by means of Aveling's apparatus, but it was interrupted by the formation of a clot in the syringe, and the patient died. In the third case, one of placenta prævia, the Resident Obstetric Assistant had to deal with the immediate danger of death from hæmorrhage in the absence of the Assistant Obstetric Physician. He adopted a plan, the use of which I have not seen elsewhere recorded, and one which does not seem advisable, except in desperate circumstances. No donor of blood could be obtained, but some of that lost by the patient had been caught in a clean vessel. About six ounces of this were strained through muslin, a little phosphate of soda added, and transfusion commenced with an Aveling's apparatus, which was the only one at hand. A clot, however, was soon formed in the vein, and extended into the tube of the syringe. The patient died about half an hour later. Of the period of twelve years included in the statistics above mentioned, the Charity was for four years under the direction of Dr. Braxton Hicks, for six years under that of Dr. Phillips, and for two years under my own.

During the interval since the last report of the Charity, published in the 'Guy's Hospital Reports' for 1876, an interval in which more than 16,000 additional deliveries are comprised, transfusion has been performed in only two other cases, the histories of which I propose to record in the present paper. One of these patients recovered and one died.

There are two reasons for this rarity of the operation of transfusion—only one operation in about 5600 deliveries. The first is that in many fatal cases of post-partum hæmorrhage there is no time for summoning the Assistant Obstetric Physician or Resident Obstetric Assistant. There would be no chance of performing transfusion unless the immediate attendants had the necessary apparatus always with them on the spot, and were qualified themselves to perform it. In several cases, indeed, death took place before even the extern attendant summoned.

The second reason is that transfusion was only performed in those cases in which it seemed certain that it was the only chance of saving the patient. In the case of post-partum hæmorrhage, patients who do not die almost immediately recover in the great majority of cases. Out of the eleven cases fatal from post-partum hæmorrhage, but in which transfusion was not performed, included in the above-mentioned statistics, there was only one in which there would have been time to fetch a transfusion apparatus. In this instance, the patient died nineteen hours after delivery, and it is probably one in which transfusion would have been of value.

When the hæmorrhage is ante-partum, and therefore usually more gradual, it appears that a woman may be more completely drained of blood without immediately fatal syncope being produced. It is in such instances that it more often happens that, although the pulse may be somewhat revived for a time by stimulants, there do not remain sufficient blood corpuscles for the maintenance of respiration, and the patient gradually fails, and dies after a considerable number of hours. These are the cases in which transfusion of blood is of most value.

It is probable that, in some cases of transfusion which have been published as brilliant successes, the recovery may have been independent of the treatment, and that occasionally the danger may even have been increased by the transfusion.

It is not impossible even that transfusion may have killed some patients who, without it, would have had a chance of recovery. For there is no doubt that transfusion of blood, or at any rate transfusion of blood performed according to several of the methods which have been adopted, is an operation of some danger to the patient, involving a risk of killing by embolism. The great difficulty of estimating the true effect of the operation in any given case will be apparent when it is remembered that eminent authorities have recommended the transfusion of animal blood into human veins, and have even described highly beneficial results from such transfusion, in various states of exhaustion. Physiological experiments have now shown that the blood corpuscles of one animal are killed and disintegrated by the serum of the blood of any other animal, and that transfusion of this kind into the veins of a depleted animal generally proves fatal. Although, therefore,

many phthisical and other patients may have survived transfusion with the blood of lambs, the true effect of the operation can hardly have been other than injurious.

During the nine years in which I had the supervision of the Guy's Hospital Lying-in Charity, I performed transfusion only three times. There was a considerable number of other cases in which I was summoned with a view to the performance of that operation, but in which I thought that the patient would recover by the aid of other treatment. All the patients but one did so recover, and in that one case the fatal result appeared to be due to neglect of directions on the part of the patient's friends. I had watched the patient for some time, and found the pulse reappear and improve; the Resident Obstetric Assistant continued the watching for two or three hours, and the extern attendant still for several hours longer. She appeared gradually to improve throughout, and at length the extern attendant left, thinking her out of danger, but giving strict injunctions that she was not to be moved. Before he had been gone half an hour the patient's friends raised her up in bed, whereupon she suddenly fainted and died. This was the only instance in which I had to regret having decided against transfusion. The other treatment employed, besides the usual administration of stimulants and liquid nourishment, was chiefly the subcutaneous injection of ether, depression of the head by removing pillows and raising the foot of the bed upon blocks, and the so-called "auto-transfusion," or bandaging the limbs with elastic bandages, in order to retain the remaining blood near the heart.

It does not appear, therefore, that more than at most about one life out of 20,000 deliveries can have been lost by omission to perform the operation of transfusion, although in institutions like the Guy's Hospital Charity post-partum hæmorrhage is probably commoner than in private practice, partly on account of the debilitated and half-starved state of many patients, and partly because the child is often born before the attendant arrives. Even such a loss as this, however, ought to be avoided if possible. Moreover, it appears not improbable that in some cases of transfusion the operation may fail to save life because it is performed too late. The great desideratum, therefore, is to find some mode of operating which is without

any risk of killing the receiver and without risk of serious injury to the donor. The operation may then justifiably be undertaken simply to make sure that a patient will recover, and before she is in such desperate condition that it is quite certain that she will die without it.

Probably the most scientific information of any which is to be found on the subject of transfusion is contained in the investigation on that subject made by Prof. Schäfer, in 1879, for the Obstetrical Society of London.¹ Prof. Schäfer recommends, as the best method of all, arterial transfusion from the dorsalis pedis artery of the donor into that of the receiver. The arteries of each are first to be exposed and separated from the sheath for about three quarters of an inch. The distal ends of the exposed portions of artery in both are then tied, ligatures are placed loosely round the upper ends also, and these upper ends are secured by spring-clips. The transfusion apparatus itself consists simply of an india-rubber tube having a glass cannula at each end. The cannula has a tapering bevelled extremity, with a groove near the end to hold the ligature. One of these glass cannulæ is tied into the artery of the donor, the other into that of the receiver, the ends of both being directed towards the heart. The clips are then opened for about a minute, or a little longer if it seems desirable. Both arteries are then to be tied just above the clips, and finally the cannulæ are to be cut out and removed together with the pieces of artery into which they are tied.

The advantages of arterial transfusion performed after this method are the following: The blood transfused is oxygenated; if any clots are formed they produce embolism only of peripheral arteries of the foot and do not kill the patient, and the arterial tension of the receiver is more immediately raised, and the tendency to syncope thereby averted, than if the blood had first to make its way from a vein.

This operation may be allowed to be theoretically the most perfect mode of transfusion. The chief obvious objection to it is that it is a somewhat serious operation for the donor, and that since it is performed on the foot it requires him to be kept quiet in bed afterwards. It is, therefore, out of the question for cases like those of the Guy's Hospital Charity, in which the husband is generally the only possible donor of blood, while

¹ 'Obstet. Trans.,' vol. xxi.

there is no other bed for him to lie upon during the wife's lying-in period, and probably he has to go to work the next day. Since one case at any rate has been recorded in which the operation of transfusion was fatal to the donor, though performed only on the arm, the necessity for proper after-care on his part is the more apparent. On account of these difficulties in the way of arterial transfusion I am not aware that the method recommended by Prof. Schäfer has yet been actually put in practice.

As the next best method Prof. Schäfer recommends direct transfusion from vein to vein through a simple elastic tube without any pumping apparatus. The tube and glass cannulæ are the same as in the former case. Both veins are to be exposed, the cannula is to be tied into the vein of the donor, but only held in that of the receiver. Blood is to be allowed to flow for about three minutes, or until the condition of the donor or that of the receiver shows that sufficient has been transfused.

With these methods suggested by Prof. Schäfer have to be compared, first, Aveling's well-known transfusion apparatus and also that of Roussel. In both of these the transfusion is from vein to vein, and there is a pump to hasten the flow. Although the first few ounces of blood generally pass quite successfully, yet, after a while, clots generally form in the tube in both apparatuses. And, therefore, although apparently very successful transfusions have been performed with both instruments, yet there is no security with either that the operation may not kill the patient by pulmonary embolism instead of curing. On this account both are condemned by Prof. Schäfer, and I am disposed to agree with this condemnation provided that a better method is found practicable.

Roussel's instrument appears indeed most successful as demonstrated before a Society. A donor of blood is provided, the vein is pricked, the blood appears at the delivering cannula, a few drachms are allowed to flow, and the operation is stopped. The really critical time, however, would be towards the end of the operation when some ounces have already flowed. Another drawback to these instruments is that such safety as can be attained by them lies solely in the operation being completed quickly. The inventor of the instrument, especially if he

practises transfusion on cases other than those of hæmorrhage, may be very rapid and dexterous in its use. But a general practitioner, having occasion to operate perhaps only once in a lifetime, might occupy rather more time, and the risk to the patient is thus immensely increased. A third objection to both instruments is, that they are made of india-rubber, that of Roussel being of some complexity. If they have been lying unused for two or three years they are apt to be found cracked and useless.

There is another method, of which so many supposed successes have been reported, and which appears to have so many advantages, that, even after hearing Prof. Schäfer's paper, I resolved still to make further trial of it. This is the plan of using defibrinated blood. It has the great advantage that the operation for the donor is that of simple venesection, that the blood may be prepared in another room, so that the donor's nerves may not be shaken by the sight of his dying wife, and that there is no need for any hurry or rapidity of procedure. The disadvantages are that the white corpuscles at any rate are more or less disintegrated during the exposure of the blood, that, even after the defibrination, other small clots may form and produce embolism, that the same result may be caused through clots produced in the recipient's blood by the liberated fibrin-ferment, and finally, that septic germs may be introduced with the blood. With regard to the reality of these dangers experience alone can decide. This method has been chiefly used in Ireland, by Dr. McDonnell, of Dublin, and others who have employed his transfusion instrument. It appears to have been successful in several instances. In one case, however, recorded by Dr. Atthill,¹ in which Dr. McDonnell himself performed the transfusion, the operation was followed by no benefit, although prior to the operation Dr. Atthill states that the case did not appear by any means hopeless. During the operation the patient's respiration became heavy and of a groaning character; the operation was followed by restlessness, dyspnœa, and complaint of great distress and pain in the chest. The patient died two hours afterwards, six hours after delivery. Although, at the autopsy, no plug was found in any of the larger branches of the pulmonary artery, Dr. Atthill considered that it was a fair

¹ 'Dublin Journ. of Med. Science,' 1877.

inference that minute emboli might have been formed. Old-standing disease at the base of the left lung existed.

The following case, in which defibrinated blood was employed for transfusion in the Guy's Charity, may afford some further evidence on the subject :

Ann D—, æt. 33, was pregnant for the fourth time. On October 20th, 1882, when pregnant about six months she had severe hæmorrhage; this was checked by opium and rest. She was advised to continue resting, but on the morning of October 26th she went out of doors against orders. Hæmorrhage came on again in consequence at 8 a.m. She did not, however, send for assistance till 6 p.m.; bleeding having continued throughout that interval. The extern attendant, on his arrival, found her completely blanched, pulseless, vomiting, and complaining of pain in the abdomen and back. The os was slightly dilated, the placenta presenting. The vagina was plugged until a Barnes' bag was fetched and introduced. As soon as the os was sufficiently dilated by means of the bag, version was performed by the Resident Obstetric Assistant, and a still-born child extracted. No more hæmorrhage than usual took place after delivery, the uterus being gently kneaded for an hour.

Four subcutaneous injections, each of thirty minims of ether, were given in succession, hot blankets and hot water bottles were applied, and eggs, milk, and brandy were administered, and not vomited. About an hour later, a small radial pulse, rate 130, could be detected. This lasted only fifty minutes, and was not felt again.

She was watched until 7 a.m. on the 27th, when she was thought to be improving, as there was no restlessness, and food was taken freely, and retained. She slept from time to time during the day, and respiration remained quiet, but no radial pulse was felt. She vomited twice during the day. In the afternoon, as she did not improve, Esmarch's elastic bandages were applied to the legs. As she had now taken a dislike to milk, Derby and Gosden's fluid meat was given.

At 6 p.m., as she appeared worse, and vomiting had recommenced, I was summoned to see the patient, with a view to the performance of transfusion, and I decided to undertake that operation. The husband declined to furnish the blood, saying

that he was himself a delicate man. The patient's mother, however, agreed to be the donor. It was decided to defibrinate the blood. This was done by stirring it gently with a fork for five minutes, the basin containing the blood being kept warm in a larger basin of warm water, and afterwards straining it through muslin. The apparatus used was simply a glass funnel, connected by an elastic tube about three feet long with the delivery cannula of an Aveling's transfusion apparatus. The funnel was kept filled from a small jug containing the blood, the jug being meanwhile kept warm in a basin of warm water. With this apparatus the blood flows into the vein simply by the force of gravity, when the funnel is raised, care being taken to make sure that the tube and cannula are quite full of blood and free from air, before the cannula is introduced into the vein. If it is desired to accelerate the flow this is easily done without any expansion in the tube or other form of pumping apparatus. All that is necessary is to seize the tube near its upper part with the finger and thumb of the left hand, and then run the finger and thumb of the right hand, previously oiled, for a few inches along the outside of the tube from the same point downward, thus forcing the blood onward. Then the tube is released at the upper point, and the same process repeated.

The amount of blood, when defibrinated, was about twelve ounces. As it flowed rather slowly from the force of gravity only, the flow was accelerated in the manner just described. No benefit appeared to result from the transfusion, and no pulse ever became perceptible at the wrist. On the other hand, there was no dyspnoea, complaint of oppression at the chest, or other symptoms indicating pulmonary embolism. The patient, however, died rather more quickly than I should have expected her to die in the absence of any transfusion, namely about an hour after the operation was completed.

It is a significant circumstance that during the transfusion a small clot was observed in the funnel and removed from it. It is thus proved that defibrination for five minutes is not sufficient to be a security against further formation of clots. Minute shreds of fibrin, sufficient to cause multiple small emboli, might easily be formed unnoticed in this or any other apparatus for the injection of defibrinated blood. It would, with this apparatus, be some additional security to line the

264 *Transfusion of Blood for Puerperal Hæmorrhage.*

funnel with a small sheet of muslin, arranged like a sheet of filtering-paper, thus filtering the blood a second time at the last moment.

It was noticed that, when the incision was made in the patient's arm to expose the vein, the blood which flowed was quite thin and pale, like pink water. This is explained by the fact that she had been able to retain liquid food in the stomach during an interval of nearly twenty-four hours since the hæmorrhage. It was clear that a considerable quantity of liquid had been absorbed from the stomach, sufficient to dilute the blood in a very manifest manner. It is evident, therefore, that she died from want of the vital constituents of the blood, and not merely from deficiency in its volume, and that no intravenous injection of saline solution would have been likely to avail.

In two other cases in which I superintended the operation of transfusion of defibrinated blood in Guy's Hospital the results were not altogether satisfactory. One of these was a case in which the physician in charge directed transfusion to be performed for diabetic coma. The operation was carried out with great facility, and the patient appeared revived for a time. He died, however, in a few days, and, at the autopsy the vein into which the injection had been made was found full of pus. This result seems to show that some septic germs were introduced with the blood, although the proneness to suppuration may have been due, in great measure, to the diabetic condition.

The other case was that of a patient under the care of Dr. Braxton Hicks. Abdominal section had been performed for extra-uterine foetation, and the result appeared for some time to be very successful. Secondary hæmorrhage, however, occurred on the separation of the placenta, more than a week after the operation, and proved fatal. In this instance the patient was in an extreme condition, and it was probably too late for any kind of transfusion to save her. She died, however, after the injection of defibrinated blood rather more rapidly than I expected her to die without it.

The issue of these cases, and especially that of the one first recorded, which was the last in point of date, led me to determine to try in future a different plan of transfusion, since I considered that the quantity of blood transfused ought to have been sufficient to save the patient, and since death appeared to

have been, if anything, accelerated rather than deferred by the operation.

There is another mode of transfusion, recommended by Dr. Braxton Hicks,¹ which has the same advantages of convenience as that of injecting defibrinated blood. This is to prevent coagulation by mixing the blood with strong solution of phosphate of soda, three ounces to the pint, one part of the solution being added to three parts of blood. To this Professor Schäfer objects that such a strong saline solution will certainly kill the blood corpuscles. And although Dr. Hicks recorded four cases in which he performed the operation with great ease and comfort, yet the patients did not recover. I have not, therefore, ventured to make trial of this method.

In the following case I used a slight modification of the second method recommended by Prof. Schäfer, with a successful result, although the patient appeared to be in fully as desperate a condition as in the case first recorded where injection of defibrinated blood failed to save life.

Ellen C—, æt. 41, was pregnant for the twelfth time. On October 20th, 1882, when pregnant about six months, she had severe hæmorrhage, and two handfuls of clot were passed. The bleeding came on quite suddenly, and the patient became much blanched. Placenta prævia was diagnosed. She was kept at rest, and treated with opium.

On November 3rd, at 9 a.m., the hæmorrhage came on again. The os was found somewhat dilated, and the placenta presenting. The vagina was plugged until a Barnes' bag could be introduced. As soon as the os was dilated enough, version was performed by the Resident Obstetric Assistant, and a still-born child delivered. No foetal heart could be heard before the performance of version. There was no hæmorrhage from the time of the insertion of the Barnes' bag until delivery, and no undue hæmorrhage afterwards.

The patient remained excessively blanched, and in a state of semi-syncope. Subcutaneous injections of brandy and ether were given. The pulse could be just felt, rate 128. She took milk, eggs, and brandy, and did not vomit.

About 5 p.m. I was summoned to consider the question of transfusion, as she had become pulseless, very restless, and with

¹ 'Guy's Hosp. Rep.,' 3rd series, vol. xiv, 1868.

cold sweat on her face. Ether injections were again given, and the legs were bandaged with Esmarch's elastic bandages. The pulse then became again perceptible, and the restlessness disappeared. Transfusion was, therefore, deferred. At 8 p.m., however, the condition had become much worse. Restlessness had returned, no pulse could be felt, and respiration was sighing and irregular, pallor extreme. Transfusion was now decided on. The husband consented to be the donor, and went through the operation with great steadiness.

The apparatus used was simply a piece of elastic tubing twelve inches long, having at its extremities the receiving and delivery cannulæ of an Aveling's syringe, but no bulb or pumping apparatus. The cannula was first inserted into the patient's vein, then the donor's vein was exposed, opened, and the receiving cannula inserted into that. As soon as blood flowed from the end of the tube, its metal terminal was inserted into the cannula already in the patient's vein. The tube was left connected for about five minutes. At the end of that time the patient was still pulseless, and no marked change was observed in the donor's pulse. The tube was, therefore, taken out from the delivery cannula, and it was found that the flow had been stopped by a clot near the end of the tube. The tube was then separated and freed from clot without much difficulty, by running the oiled finger and thumb down the outside of it in the manner previously described. A finger was meanwhile placed upon the receiving cannula to stop the flow of blood. The tube with delivery cannula was then again joined to the receiving cannula and, as soon as blood flowed from the delivery cannula, this was again inserted into the vein. After a few minutes, the flow of blood in the receiver's vein appeared to be again arrested. The tube was therefore a second time cleared of clot in the same way as before, and again connected. The flow eventually became stopped for the third time. There was no perceptible improvement in the patient, but as the donor's pulse had become rather rapid no attempt was made to renew the flow.

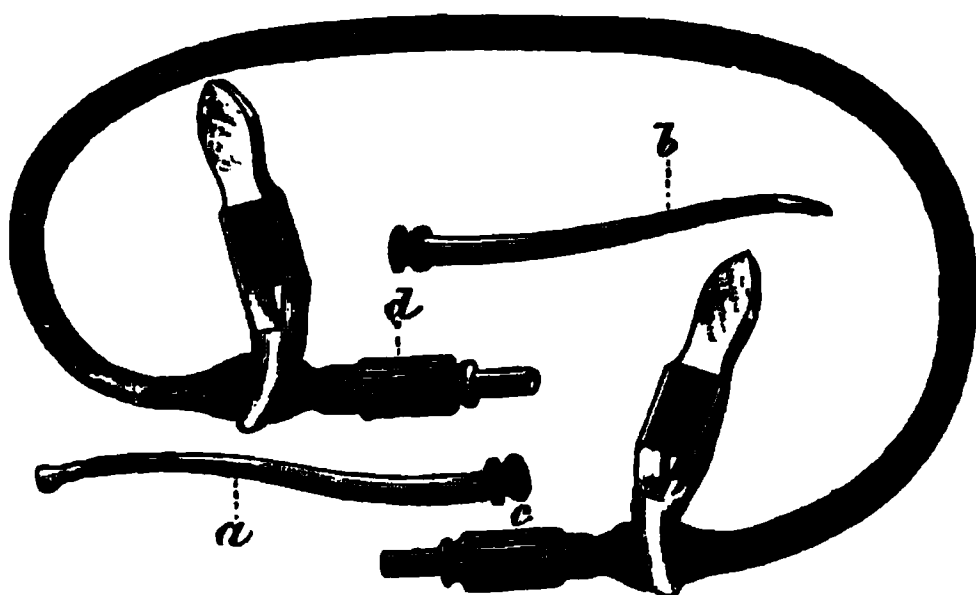
I completed the operation under the impression that little good had been effected, and that the patient would certainly die. From that time, however, she improved steadily though very slowly. No food was given until 8 a.m. on account of the

vomiting, but from that time the vomiting ceased and she was able to take small quantities. After 5 a.m. the pulse rapidly improved.

She remained excessively blanched and weak for many weeks. On November 15th phlegmasia dolens appeared in the left leg, and on the 28th in the right. After a long illness she completely recovered.

I am disposed, in any future case, to adopt the same method as that used in this, for it appears to be clear that it has the very great and what I consider to be the most essential advantage of all, namely, that although a clot may be formed in the tube it is not carried into the receiver's circulation. The operation may, therefore, be considered practically without risk or almost without risk of doing injury to the patient. It may, therefore, I think justifiably be performed even before that stage is reached at which it becomes certain that she will not recover without transfusion.

The plan which I used differs from that of Prof. Schäfer in the use of Aveling's terminals and cannulæ instead of glass cannulæ, which have to be tied into the veins. In this way the operation is rendered somewhat simpler and shorter. There is another advantage, namely, that it is possible at any moment to ascertain whether the flow is still going on freely by taking the terminal (*d*) (see Fig.) for a moment out of the delivery



cannula (*b*), and replacing it if all is right. Also, if the tube should be blocked by clot, it can be cleared by the method used in the operation described without interfering with the receiving cannula in the donor's vein, and the process set going afresh.

I think it better to dispense with the taps with which the metal terminals fitting into the cannulæ are provided in Aveling's apparatus, and to use instead spring-clips on the outside of the tube. For if the tap is not turned with perfect exactitude in the axis of the tube, a projection is made on the inside of the tube which may form a starting-point for clotting. The terminals which I recommend are shown in the figure (*c*, *d*). They are perfectly smooth inside, and the ends are made as thin as possible, so that there is scarcely any projecting rim within the lumen of the tube when all is united either on the side of the elastic tube or on that of the metal cannula. The outside of the terminal has a grooved projection which is easily held by the finger and thumb in inserting the terminal into the cannula or drawing it out. This arrangement has certain advantages compared with more elaborate instruments. It occupies scarcely any room, and may without inconvenience be carried constantly in the obstetric bag. The only part made of india-rubber is the elastic tube. This may be renewed from time to time without appreciable cost, and then there is no danger of finding the apparatus cracked and useless at the moment of need. Or, if necessary, the tube can always be replaced with a piece of ordinary drainage-tube.

With this apparatus the mode of procedure should be as follows: Place the transfusion tube, including terminals (see Fig.) and cannulæ in a hot solution of common salt or carbonate of soda (gr. lx ad Oj). When the tube is full and all air removed from it place a spring-clip on it at each end close to the terminal. Tie tapes round the arm of the receiver, first above and then below the vein which is to be opened. Prepare the vein by exposing a portion of it and passing a probe underneath it. Then tie tapes round the arm of the donor, first above and then below the point where the vein is to be opened. Expose the vein and pass a probe underneath it. Now let the donor sit by the bedside and place his arm close to that of the patient. Take the delivery cannula (*b*) out of the saline solution, open the receiver's vein by a snip with sharp-pointed scissors, and see that the cannula slips readily into it. Removing the cannula, pass a small director into the vein that the opening may not be lost, and remove the tape above the opening.

Take the transfusion-tube with both cannulæ affixed, open

the donor's vein by a snip with scissors, and slip the receiving cannula (*a*) into it, passing it gently on so far that by its conical shape it fills the vein, or the opening in the vein, and does not allow blood to escape by the side. Let an assistant hold the cannula in place, remove the lower tape from the donor's arm and remove the spring clips, keeping the delivery cannula slightly raised above the donor's vein. As soon as blood begins to flow from the delivery cannula slip the cannula into the receiver's vein and hold it there, having passed it in far enough to prevent escape of blood by the side as in the case of the receiving cannula. The flow will be aided if the receiver's arm is raised on a pillow slightly above the level of the shoulder.

With this method the quantity of blood transfused cannot be measured. We must judge when to conclude the operation partly by the time of the flow, which should not be less than about five minutes, and partly by the effect on the pulses of the donor and receiver. When the cannulae are withdrawn the remaining tapes are removed and the veins closed by a pad and bandage in the usual way.

If no donor of blood could be obtained except a highly nervous woman, or a greatly excited husband, whom I thought able to bear a simple venesection but not to go steadily through the operation of direct transfusion, and if the patient seemed certain to die without transfusion, I should still adopt the plan of injecting defibrinated blood, although I consider it not without risk of killing by embolism.

For this operation the terminal (*d*) and delivery cannula (*b*) of the same apparatus may be used, fitted to an elastic tube about three feet long and not less than three sixteenths of an inch in calibre. The other end of the tube is attached to a glass funnel. The mode of procedure will then be as follows: First expose the receiver's vein and place a probe under it, then draw, defibrinate, and filter the blood through muslin, place it in a small jug which is kept warm in a basin of warm water, place a spring-clip at the end of the transfusion-tube close to the terminal, hold the tube vertical with the funnel uppermost and fill the funnel with hot solution of common salt or carbonate of soda (gr. lx ad Oj) previously prepared, open the spring-clip and let the solution run out till the funnel is just empty and the tube alone full, then close the clip again. Now pour the blood into the funnel,

open the clip till the blood begins to escape from the cannula and then close it again, open the receiver's vein, and slip the cannula into it, keeping the arm somewhat elevated on a pillow above the level of the shoulder. When the clip is taken off and the funnel raised the blood will generally flow in by the force of gravity. The funnel must of course be kept replenished as the level of the blood in it falls. If necessary the flow may be accelerated by running the oiled finger and thumb down the tube. But if the flow seems to be arrested or nearly arrested, it is better first to withdraw the cannula for a moment from the vein, and make sure that the flow is not stopped by a clot in the cannula or tube. I consider this simple arrangement of funnel and tube equal, and even superior, in its action to any more complicated india-rubber apparatus, which is apt to be found unfit for use when wanted unexpectedly.

The same arrangement of funnel and tube may be used very conveniently for the injection of saline solutions. Such saline solutions are certainly inferior in efficacy to the transfusion of blood. They may avert fatal syncope following shortly after sudden hæmorrhage, such as post-partum hæmorrhage, and may be used when blood cannot be obtained. In cases like those now recorded, when life is prolonged for a considerable number of hours, and fluid is meanwhile freely absorbed from the stomach, they would probably prove entirely useless. Even when used for the immediate effect of sudden hæmorrhage the benefit derived from them has, in several instances, proved only temporary.

**CASE OF TYPHOID FEVER,
FATAL ON THE SEVENTY-SIXTH DAY,
WITH HÆMORRHAGE DUE TO RECENT TYPHOUS
ULCERATION IN THE ILEUM.**

BY SIR W. W. GULL, BART., M.D.

It would seem probable that the typhous process, once begun, has to run a course longer or shorter until the body becomes insusceptible to its influence. The old and current opinion, still more or less entertained, is that morbid processes, set going by animal poisons, are put an end to by the elimination of the poison which causes them.

This view does not seem to be supported by a consideration of all the facts. For instance, an attack of smallpox has two sets of phenomena; the physiological disturbances of the acute attack, which end in a given time; and the underlying process, which is permanent, and which shows itself by the insusceptibility of the patient, for the most part, to a second attack. The physiological disturbances—which are called the disease—namely, the quick pulse, high temperature, delirium, &c., end, and the case takes a favorable course, when the patient has fully generated the poison; and is most dangerous on that account to other persons, whilst at the same time he obtains an immunity for himself.

I believe, therefore, that the former teaching at Guy's is confirmed by the further progress of pathological knowledge,

viz. that the symptoms set up by animal-fever poisons end when the process of *assimilation* is complete, and that the process of *elimination* has nothing whatever to do with recovery.

If this be the case we should expect, as we find, great variety in the intensity of the symptoms during this process of assimilation.

Vaccination has enabled us to get the assimilative process of smallpox with little or no physiological disturbance. In respect of typhoid fever, the physiological disturbances are extremely variable, often so doubtful as to be hardly distinguishable; at other times there is much delirium, high temperature, rapid pulse, acute bronchial symptoms, &c, &c.

Further, in typhoid the assimilative process is not always completed in one attack, say of twenty-eight days, but often this process is repeated again and again, constituting a *double*, or *triple* or even *quadruple typhoid*. When fifty-six days are completed there may be a *third* repetition, or, as in the case I have here to record, probably a *fourth*.

Mr. E. P—, æt. 37, consulted his medical attendant, Sept. 14th, 1879, for diarrhœa and headache of a few days' standing. Tongue coated; temp. 102°. For two weeks the symptoms had a mild course of ordinary typhoid, and the patient seemed to be convalescing.

October 9th.—Return of headache; temperature again rising to 102°, with some sweating; no diarrhœa, bowels rather inactive.

The course of the case was favorable, and at the end of the month he was thought to be convalescent, and on November 3rd all the symptoms had apparently subsided.

November 7th.—Return of headache; no diarrhœa, on the contrary, bowels inactive; supposed to be relapse of typhoid symptoms.

Patient went on favorably till November 18th, when febrile symptoms increased. Temp. 103·2°, pulse 108.

21st.—Temp. 104·2°, pulse 120; delirium, tympanitic abdomen, intestinal hæmorrhage, and death the following day.

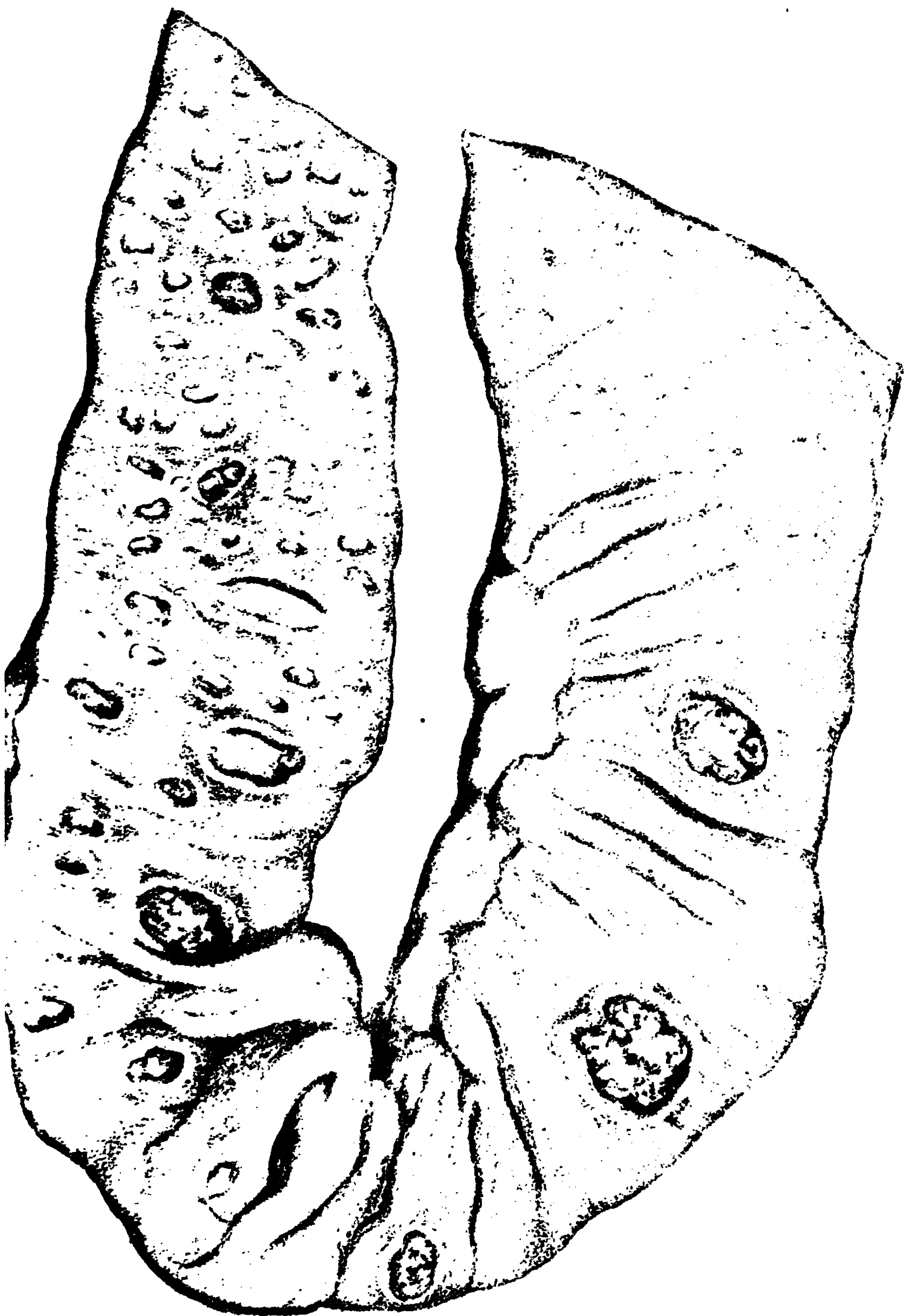
On removing the ileum it presented various stages of recent ulceration and old healing ulcers. Dr. Goodhart has kindly given the following account of it:

“The lower part of the ileum was excessively diseased. The worst part is not represented in the drawing, as the mucous membrane was in a general state of sloughing. Just above this there were distinct ulcers and fleshy swelling of the mucous membrane, which seemed to me to indicate certainly three, and possibly four, stages of the disease. Thus, at the lower part, were ulcers with slough separated, and a distinct cicatrisation at the edges; the margins being adherent to the sore and skinning over. There were others in which the slough had cleanly separated, and the muscular fibre of the bowel formed the floor, but as yet no healing had taken place (these are probably co-eval with those just mentioned, but of more sluggish process); others in which the yellow typhoid slough was still separating; others with the solitary follicles and parts of the agminate glands swollen, and with small, ragged, granular ulcers in the centres of the diseased parts; and, lastly, there was a condition of general early swelling of the mucous membrane. The severity of the ulcerative process was as usual expended upon that part of the ileum next to the ileo-cæcal valve, but even those ulcers at the upper part, and therefore presumably those last affected, showed two distinct stages—one of ulceration with the slough cleared away, and recent swelling of the remainder of the patch.”

DESCRIPTION OF PLATE

Illustrating Sir W. W. Gull's Case of Typhoid Fever.

Lower portion of the ileum, showing recent typhous ulcerations from one of which recent bleeding had occurred; also ulcerations in various stages, some even healed, and the whole indicating the effects of the typhous process over seventy-six days.



CASES ILLUSTRATING THE VARIOUS RESULTS
OF
PARTIAL SUBCUTANEOUS LACERATION
OF ARTERIES.

BY CHARTERS J. SYMONDS, M.S.

ALL the following cases have occurred recently in the practice of this hospital, and for most of them I am indebted to my colleagues. Two cases (Jupe and Kellher) were under the care of Mr. Cooper Forster, Kellher passing later under the care of Mr. Davies-Colley; one (Stiff) under Mr. Bryant; two (Turner and Donelly) under Mr. Davies-Colley; the remaining two cases (Schultz and Littlewood) were under my own care.

Of the various injuries sustained by the large arteries, without accompanying wound of the skin, viz. complete laceration, partial laceration, and puncture from a spicule of bone, I propose to confine my attention to the second form.

Meaning of the term, and nature of injury.—By partial laceration is understood an injury in which the internal tunics alone are ruptured, the external, as in ligation, remaining intact. Under such a heading might also be included laceration where all the coats are divided and the lumen opened, but in which some portion of the wall remains continuous. Such an injury would manifestly give rise when extensive to the same signs as complete laceration, and when slight to the

signs accompanying puncture of an artery as by a spicule of bone.

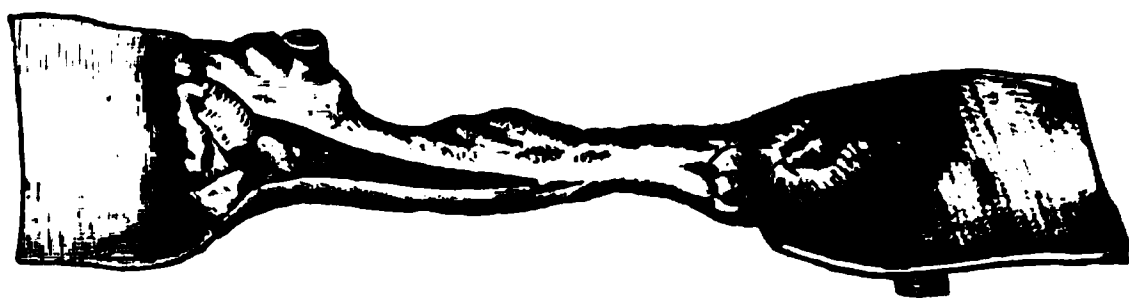
The present cases illustrate that form of injury in which there is no escape of blood from the vessel, the immediate swelling that does occur being due to the escape of blood from the small vessels in the over-lying and surrounding tissues.

Mr. Poland, in his paper on "Rupture¹ of the Popliteal Artery and Popliteal Aneurism" in an earlier number of these 'Reports,' refers to the form of injury here considered in the following terms:—"The popliteal artery is very liable to incomplete rupture of one or more of its coats, such as is so frequently observed in the formation of popliteal aneurism, in many instances of which the lesion may be distinctly traced to external injury or violent exertion and straining of the knee." He dismisses this injury, however, for, as he states, he is dealing only with "lacerated or ruptured artery with extravasation."

The laceration may extend all round the vessel so as to divide entirely the two inner coats. Then they curl up and occlude the vessel, or, as the second specimen referred to shows, there may be little or no incurvation. These appearances are beautifully illustrated in Mr. Bryant's work on surgery,² and, as has been observed, resemble the effect produced by a ligature or torsion.

Of the cases narrated below one only was fatal (Kellher). The accompanying woodcut shows the appearance of the prepara-

Fig. 1.



tion now in our museum.³ At the time of the inspection the then Registrar, Mr. F. Durham, wrote of the condition of the artery as follows: "The common femoral artery was severely bruised for two inches, but was not divided. On opening the artery the internal and middle coats were found lacerated,

¹ Series iii, vol. vi.

² 'Practice of Surgery,' 3rd ed., vol. i, p. 412.

³ 1504th.

separated from the external, and rolled up inwards, completely occluding the vessel."

The museum catalogue and the post-mortem report describe the specimen as a part of the external iliac, and from the arrangement of the branches arising from the trunk this appears to be the case. As the drawing shows, the two inner coats on the distal side of the rupture are twisted and curled up, and did in the recent date probably quite fill the vessel. The proximal end of the rupture shows less incurving, but there seems to be sufficient with the accompanying coagulation of blood to occlude the vessel. Rather more than an inch intervenes between the ends, which are connected by the external coat. This coat as will be seen forms a distinct tube. The drawings in Mr. Bryant's work also show one end (distal) to be curled up and twisted much more than the other.

There is another preparation¹ in the museum of a popliteal artery showing the same injury. It was removed from a man, æt. 36, admitted under Mr. Poland, October 2nd, 1863, having been run over two or three days before. He died on the 5th. The left leg was much injured, and there was a wound which at the inspection was found to enter the knee. The leg was swollen, and gangrene had already commenced when admitted. "There was much exudation of dirty fluid around the knee-joint. The popliteal artery was exposed, and it was found not to be ruptured externally, nor was any blood effused around it, but it was observed to be contracted for about an inch of its length. On removing the vessel and opening it, it was seen to have undergone a transverse laceration, by which its inner and middle coats were completely separated, but leaving the external cellular coat entire. This had contracted on itself to the smallest size, and was full of a firm coagulum. The femoral for some distance above was full of firm clot. The separation of the two ends of the vessel was to the extent of about an inch." The above is from a report made by Dr. Wilks in the post-mortem records for 1863. The preparation shows the proximal end to contain a coagulum, while the distal one is patent. There is no evidence of incurvation of the inner coats at either end, a condition differing from that usually described.

Nélaton, in describing Bérard's case mentioned below, says,¹ "A rupture of the two internal tunics of the axillary artery all round the circumference had occurred. The cellular tissue had become elongated like a tube."²

It appears from the study of the cases here recorded that besides the laceration above referred to, which extends all round the vessel, there is a slighter form in which but a part of the wall is injured, for it will be observed on reading these cases (Donelly, Turner, and Jupe) that pulsation returned after a short time—five, three, and seven days respectively. This seems too short a time for the establishment of collateral circulation. In Sir James Paget's³ case of ligature of the axillary the pulse was not counted till five weeks after the operation. In what way the rapid return of pulsation may be explained will be considered later on.

The form of violence.—These cases illustrate two distinct ways in which the laceration is produced.

First, by a direct blow. In Stiff's case a cart-wheel passed over the groin and contused the common femoral just below Poupart's ligament. The injury was confined to the artery and the overlying structures, and so slight was the man's pain that to him the leg was in no way different from the other. Again, in Littlewood's case the temporal artery was struck by the corner of a box. In Kellher's case, also, the rupture of the artery was caused by a cart-wheel.

Secondly, by overstretching. This form of injury is illustrated by the case of Schultz. The arm was entangled in a rope connected with the revolving drum of a steam winch. The man was suddenly dragged forward and turned over the drum. The subclavian artery was ruptured in this case, and, as will be seen on reading the report, the cords of the brachial plexus were at the same time torn through.

Thirdly, by a combination of contusion and stretching. This injury occurs in dislocations of the shoulder where the head of

¹ Nélaton, tome ii, p. 368.

² Two interesting cases will be found recorded in the 'Berliner klin. Woch.,' No. 15, 1884. In one the brachial, and in the other the popliteal, was injured. In both gangrene occurred, and amputation became necessary. The injury to the brachial was limited to a transverse rupture of the intima, 3 mm. in extent, and this coat was not further detached. A coagulum obstructed the vessel.

³ 'St. Bartholomew's Hospital Reports,' vol. ii, p. 106.

the bone is driven against the artery. A case (of Bérard's) is recorded by Nélaton of a subcoracoid displacement in which pulsation in the vessels of the arm was absent. The axillary was obliterated by the method above mentioned, gangrene of several fingers ensued, and the patient died.

Nélaton records also a case of his own of subglenoid dislocation, in which he says the two internal tunics were torn through and a "false consecutive aneurysm" ensued.

Both these cases are mentioned by the late Mr. Callender,¹ who also records an instance occurring in the practice of Mr. Stanley. This was supposed to be a partial dislocation of the humerus. There was no pulse at the wrist. Many years afterwards the patient died, and Mr. Stanley found on dissection "that there had been a fracture through the anatomical neck of the humerus, with obliteration of the axillary artery opposite the broken part of the bone." Mr. Callender suggests that only the inner and middle coats had given way probably at the moment of the accident. This view seems to explain the case fully.

The effects on the artery and the circulation.—Several results appear to follow this form of injury and call for some remarks.

The *first* and most frequent effect is *complete occlusion* of the vessel and arrest of the circulation. The cases of Stiff, Kellher, and Schultz are good examples. The arrest of the circulation was in neither of the two surviving cases followed by gangrene, but that this does occur is proved by Bérard's case of ruptured axillary. The occurrence of gangrene depends not only upon the vessel injured, but also, and largely, upon the amount of injury sustained by the surrounding parts. Mr. Holmes² remarks on this point, "If the injury be uncomplicated it probably will not lead to gangrene." There are not sufficient particulars of Bérard's case to enable one to say whether other structures were injured or not. It is probable also that in elderly people there is a greater liability to gangrene on account of the senile changes in the vessels.

Nothing could have been neater than the closure of the common femoral in Stiff's case. He had far less disturbance of circulation than usually follows ligature of the superficial

¹ 'St. Bartholomew's Hospital Reports,' vol. ii, p. 103.

² 'System of Surgery,' vol. i, p. 360.

femoral, he was allowed to walk in about six weeks, and no doubt would have been up earlier had he not been under good supervision, for the man felt his leg to be as good as the other. There never was any oedema, and at the present time he is strong and well.

The *second* effect is *temporary arrest* of circulation. Three of the cases exhibit this result. In one case, Jupe's, the obstruction was higher than the third part of the subclavian, in both the others the brachial was injured. In one of the latter (Donelly) the pulsation ceased abruptly at the middle of the arm immediately after the accident, and yet on the fifth day the radial pulsated.

In these cases one can only theorise and suppose that a very limited laceration takes place, sufficient with the attendant clotting to produce arrest of the circulation, and that either by absorption or tunnelling¹ of this clot the stream again passes on, the laceration healing so as to prevent the formation of aneurysm. It may be here observed that the large vessels of a limb may contain plenty of blood without pulsation existing. The presence of blood I have been able to demonstrate by the production of a wave in the radial, obtained by compressing the brachial and suddenly removing the pressure. This was observed in Jupe's case and also in Schultz. In the latter, on amputating his arm there was free bleeding from the brachial. I mention this to suggest that possibly in these so-called temporary occlusions there may all the time be a gentle stream traversing the vessel, insufficient to produce a pulse, but capable of being recognised by the above method. In Jupe's case we felt this wave on the third day, the first time the observation was made. This was accidentally discovered, one finger happening to be on the radial while the other hand was holding the arm. On relaxing the grasp a pulsation was felt. It is possible we might have found this wave on the first day.

If it be true that there is not complete arrest even temporarily one can understand the force of the current being sufficient to restore the channel.

A *third* and very interesting effect is the formation of an *aneurysm*.

¹ No. 1506⁴⁰ in the Guy's Museum is a femoral artery containing such a coagulum.

This ensued in one of the cases related further on (Littlewood) the artery injured being the temporal. The pulsatile swelling was observed two days after a severe blow from the corner of a box, and following so closely upon the injury leaves no doubt as to its traumatic origin. The question of interest is the exact manner in which the aneurysm arises. Assuming the external tunic to have remained entire two methods suggest themselves; the first, that rupture of the internal and middle coats occurs at the time of the accident, and that this injury involves but a part of the circumference. The circulation through the vessel is not interrupted and the laceration remaining unhealed the external coat yields to the force of the ordinary blood-pressure, and a sac is formed, at first by this coat and subsequently in the external tissues after the manner common in traumatic aneurysm.

In Nélaton's account of his case of ruptured axillary artery mentioned above, he says that the two internal tunics were torn through over a very limited extent, and that a "false consecutive aneurysm" rapidly developed. This surgeon was obliged to ligature the subclavian three months later, but the aneurysm burst and the patient died.

This is the explanation usually offered when aneurysm follows an injury to an artery where no wound has been inflicted, and this whether the injury be a direct blow, as in the instance under consideration, or an overstretching of the vessel.

Writing on this subject Lidell¹ says, "When in such cases of incomplete laceration of arteries, the incurvation of the internal and middle coats does not suffice to block up the channel of the injured vessel, the pressure of the blood is liable to stretch the outer coat at the place of injury and expand it into an aneurysmal sac."

While adopting this explanation as the most probable it must be remembered that it is largely a matter of inference.

In Mr. Pick's case of femoral aneurysm,² quoted also by Lidell, it is stated that the man sustained a rupture of the internal and middle coats, and that the external coat gradually gave way. This was the case of a policeman, æt. 31, who fell upon his left knee and then backwards over his prisoner. "The

¹ 'Ashurst's Encyclopædia of Surgery,' vol. iii, p. 144.

² 'St. George's Hospital Reports,' vol. vi, p. 161.

nature of the injury," says Mr. Pick, "was such as would be likely to produce a severe strain on the coats of the artery, and produce a partial or complete rupture."

This accident occurred rather more than six months before the aneurysm was noticed. During the whole of this time numbness with varying pain existed. This man died, and at the inspection the exact relation of the coats could not be examined, on account of the hurry with which it had to be made. So that in this, which I select as a type of those upon which this view is founded, there is wanting a distinct anatomical proof of the mode of production of the aneurysm.

Mr. Holmes¹ says, "The laceration of the inner coat of an artery, instead of being repaired, is sometimes followed by the formation of an aneurysm," and then, he adds, what seems to me truly to represent our knowledge upon this mode of origin of aneurysm, that "possibly many aneurysms which are attributed to previous injuries, arise in this manner, though the subsequent changes at the part obscure their mode of origin."

The case related here offered a good opportunity of deciding as to this mode of origin. Unfortunately the patient could not, on account of her employment, submit herself to treatment, so that the further development of the case is for the present lost.

The other mode of origin which suggests itself is that an inflammatory softening follows the injury. It appears quite possible that instead of the coats actually giving way a subacute inflammation may be set up, which by softening the wall would permit dilatation and aneurysm.

It is well known that hæmorrhage occurs some days after bullet injuries, from inflammatory action ensuing in the arterial wall. "Such a result," says Mr. Bryant,² "is rare in civil practice," but he speaks of a "secondary subcutaneous hæmorrhage" occurring "some days after the injury." If hæmorrhage may result, aneurysm seems equally possible.

As this bears upon the general question of the causation of aneurysm, more especially of the popliteal, it is necessary to be careful in admitting injury as the cause. Attention is often first directed to a part on account of an injury which was quite

¹ 'System of Surgery,' vol. i, p. 868.

² Loc. cit., p. 413.

inadequate to lacerate the vessel. At the same time it would be difficult to say what is the smallest force necessary to rupture an atheromatous vessel.

Diagnosis and Treatment.—Loss of pulsation in the parts below the injury, with only a limited amount of swelling at the seat of rupture, and this without any pulsation, seem to be points sufficient for diagnostic purposes.

In a simple uncomplicated injury, no treatment other than rest, and careful bandaging of the limb, previously swathed in cotton wool, is necessary. The aneurysm which may follow on the large arteries is best treated, I imagine, by the ordinary methods, especially if there is reason to believe that atheroma exists. In my own case, as the artery was the temporal and the patient young, I intended first to try the effect of direct pressure, and failing this to tie the vessel on either side of the sac and remove the aneurysm.

Should the injury be recognised in a recent dislocation of the shoulder the reduction would not, I imagine, be delayed, but special precautions would have to be taken for the protection of the artery. If the dislocation be of long standing, there would be great danger of producing a complete rupture, as the vessel would most likely be adherent to the tissues surrounding the bone.

The cases which follow are arranged under three heads:

1. Those in which complete occlusion occurred.
2. Those in which temporary occlusion occurred.
3. That in which a consecutive aneurysm occurred.

Cases of complete occlusion.

CASE 1. *Rupture of the external iliac artery with complete occlusion; fractured pelvis; death.*—John Kellher, æt. 20, labourer, admitted into Guy's Hospital under Mr. Davies-Colley, for Mr. Cooper Forster, July 17th, 1876. Three quarters of an hour before, he was squeezed between the buffers of two railway carriages.

There is a swelling five inches and a half by five, rather elastic, and non-pulsating in the right groin. There is no pulsation in the femoral artery. The foot on this side is

blanched, and colder than the other. He complains of a numbing pain in the injured leg, and seems to have diminished sensation below the knee with some pain. There is a large wound in the perineum, through which the finger can be passed into the pelvis round the symphysis. No fracture of the pelvis was made out at this time. The man was much collapsed, and the examination was therefore not pushed very far.

The same night at 11 p.m., no urine having been passed, perineal section was performed by Mr. Davies-Colley. The urethra was found to be ruptured, the proximal end discovered, and a No. 10 catheter introduced.

July 18th.—Pulse 130, temp. 100°, resp. 26. As no urine had passed by the catheter some warm acidulated water was injected, on the supposition that the catheter was blocked. Towards evening, no urine having escaped, the catheter was removed, and was found free. He died early in the morning of the 19th, less than forty-eight hours after the accident.

At the autopsy a fracture was found running through the ilio-pectineal eminence into the obturator foramen, and thence through the ramus of the ischium. The bones were separated also at the symphysis. The bladder was torn at the upper and back part through all but the peritoneal coat. The urethra was ruptured one inch in front of the prostate. The rectum was uninjured, though when admitted a large clot was removed from it. A full account of the artery is given at p. 276.

CASE 2. Rupture of the inner and middle coats of the common femoral; complete occlusion; recovery with perfectly useful limb.—Thomas Stiff, æt. 35, a labourer, was admitted into Accident ward, under Mr. Bryant, May 20th, 1880, in a state of collapse and insensibility, having been run over by a loaded coal-van. The accident occurred between four and five in the afternoon. The man remained totally unconscious of his condition and whereabouts till the following morning. When visited about ten o'clock he was perfectly sensible, so that the insensibility was probably not due to any head injury.

May 21st.—The cartwheel passed over the right groin, and here, just below Poupert's ligament, is a soft elastic swelling, into which the finger can easily be sunk without any feeling of

crepitation and causing but little pain. This swelling is just over the commencement of the femoral artery, and is about one inch and a half across. There is no pulsation in it. The swelling is strictly localised, the hand passing deeply into the iliac fossa above it, but below this point there is total loss of pulsation in the arteries of the limb. The leg is rather colder than the left to one's hand; he can move his toes and ankle freely without pain, and except for a little stiffness in the knee and the injury to the groin the leg feels to the patient in no way different from the uninjured side. Sensation also is perfect. There is no sign of fracture of the pelvis. The man expressed himself as feeling very well, and seemed somewhat surprised at the amount of interest taken in his leg, which to him appeared quite sound. There is a small superficial wound just below the anterior superior spine. The limb was wrapped in cotton-wool, and he was ordered to remain in bed.

22nd.—There is no increase in the swelling. The external iliac artery cannot be felt above the swelling, but this appears due to difficulty in reaching it, for neither can the same artery be readily felt on the sound side.

23rd.—The swelling is subsiding, is still pulseless. The limb is warm, and there is no numbness or œdema. It feels to him in no way different from the other.

June 1st.—The swelling now is circumscribed and hard and is decreasing in size.

23rd.—There is a note by the ward clerk that on this day a pulse was felt in the anterior and posterior tibial arteries, and that the beats were counted, but on July 6th Mr. Bryant thought he could feel the anterior but not the posterior tibial. My own note, entered upon his discharge on July 20th, says, "He uses a stick, having given up crutches which he used for some days. There is still a hard lump in the groin without pulsation. There is no pulsation in the arteries of the limb, though it feels to him perfectly natural, but weak. There is no œdema."

May 30th, 1884.—After the lapse of just four years I have had the opportunity to-day of examining this patient. For three months after leaving the hospital he did no hard work. Since that time he has worked more or less as a labourer. He finds that the injured leg is weak when he carries a

heavy load, but this is the only inconvenience from which he suffers.

On examination the left leg is rather smaller than the right, measuring three eighths of an inch less round the calf, and one inch and three eighths less round the thigh. The nutrition of the limb is good, sensation is perfect, and there is no œdema, nor has there ever been any. A large vessel is felt pulsating just below Poupart's ligament in the situation of the common femoral, but it is not more than a fifth as strong as that on the right side. This pulsation cannot be traced for any distance down the thigh. Several smaller vessels can be felt pulsating around this spot, and are evidently enlarged collaterals. The chief of these is between the situation of the original femoral and the anterior superior spine of the ilium; another is felt in the lower part of the abdominal wall, and appears to be the superficial epigastric. No large vessels are to be felt in the gluteal region. The posterior tibial can be seen pulsating strongly, quite as powerfully as on the other side, but the pulsation in the anterior tibial is only just perceptible. On making compression over the line of the femoral at Poupart's ligament, and where the chief pulsation is to be felt, no alteration is perceptible in the posterior tibial. On compressing the collateral vessels, however, internal to the spinous process the pulsation is diminished, and quite arrested when deep pressure is made about two inches below Poupart's ligament or about the level of the profunda femoris. Compression of the popliteal also arrests the circulation. It may be added that there is no swelling at the site of rupture, but rather a loss of fulness.

The conclusion appears to be that the circulation is carried on, mainly through the branches of the internal iliac and partly by branches from the external iliac.

In describing this as a case of ruptured common femoral, it is not intended to dogmatise as to the condition of the external iliac, as it is impossible to say how far up the injury extends.

CASE 3. *Rupture and complete occlusion of the subclavian artery. Rupture of brachial plexus.*—P. Schultz, æt. 27, on September 27th, 1882, got his left arm entangled in a winch.

He was suddenly and violently dragged over the drum and crushed by the chain.

He was much collapsed and bruised over the shoulder and arm. No fracture existed. When seen soon afterwards in the London Hospital there was no pulse in any of the arteries of the left upper extremity and there was besides complete loss of motion and sensation, for besides the occlusion of the subclavian he had sustained a rupture of the brachial plexus.¹

This man came first under my notice in August, 1883, when the condition of the vessels was the same as at the present time. Examined on May 6th, 1884, twenty months after the accident, the following conditions were found: The arm is perfectly helpless. It is colder than the opposite limb and the hand is bluish and losing its wrinkles.

The supraclavicular region presents no abnormal appearance, nor is any fulness perceptible. The subclavian artery cannot be felt by the touch nor can any pulsation be discovered. The opposite artery is very easily felt as it rises rather high in the neck. Crossing the space the transversalis colli can be felt somewhat larger than that on the right side. No pulsation exists in axillary, brachial or radial. On two recent occasions, however, I was able to count a pulse in the lower half of the brachial artery which corresponded in number of beats and in rhythm with the uninjured side.

On compressing the brachial and suddenly relaxing pressure a distinct wave can be felt passing along the radial artery as was noted in Jupe's case.

The artery appears to have been injured just beyond the point where the vessels proceed from the first part, for the transversalis colli is pervious, and no part of the subclavian can be felt. The carotid pulsates normally.

Admitting this, the next difficulty is to explain how the artery was injured.

The man himself says that he was violently pulled forward and turned over the drum so that he practically stood on his head. It is to be noted at the same time that when admitted into the London Hospital there was a good deal of bruising over the shoulder, a condition which suggests direct injury.

¹ This injury will be found reported in detail in the 'Clinical Society's Transactions' for 1884.

The clavicle was not broken, but the man says that there was a swelling above this bone, which might have been due to blood-clot around the injured vessel. The most reasonable view, it seems to me, to take is that the vessel was torn by over-extension upwards and outwards, the two inner coats giving way, together with the brachial plexus. The man as I have said came first under my notice eleven months after the accident, at which time there was no lump to be felt in the line of the subclavian artery to indicate the site of injury. Mr. Banks¹ found in his case on dissecting the subclavian triangle with a view to suture of the ruptured brachial plexus, a hard lump in connection with the third part of the artery. This was three months after the accident. In the case of Stiff above narrated a swelling at first soft and later becoming hard was felt at the site of injury. It may, therefore, be assumed that the swelling had been absorbed, before the man (Schultz) came under my notice.

The non-return of pulsation may in part be due to the want of muscular power, the total activity of the limb being thus impaired. There was no pulsation felt in the case recorded by Mr. Banks.

On the 18th of May, 1884, I removed the arm just below the insertion of the deltoid, making equal lateral flaps.

The brachial artery bled freely, the blood rushing out, but with only a very slight pulsatile movement. The vessel was taken up before the division of the muscles, as there was nothing to be gained by compressing the subclavian. Several smaller muscular branches required ligature. The brachial was apparently normal in size and structure.

Cases of temporary occlusion.

CASE 4. Injury to subclavian. Partial or temporary occlusion.
—Alfred Jupe, æt. 26, railway porter.

June 18th, 1879, while shunting trucks he was caught between the buffers of two of the carriages, each of which contained ten tons of coals. He was struck over the right side of

¹ 'Trans. International Med. Congress,' vol. ii, p. 443.

the neck and chest. The accident occurred at 8.20 a.m., and he was admitted at 9 o'clock.

The man noticed that immediately after the accident the right arm felt numb and cold and was powerless.

On admission into Luke Ward under Mr. Cooper Forster the man was breathing quickly, 36 to the minute, and could only lie on the right side with comfort. He was not suffering from collapse, but, like most men who have sustained a "buffer accident," he was very much frightened.

There was a bruise over the left pectoral region, and a graze running across the front of the chest over the upper part of the sternum.

The skin over the left scapular region was bruised and grazed, and there were similar injuries on the left wrist and right forearm. The line of junction between the first and second pieces of the sternum is somewhat prominent, but there is no movement. The right clavicle is more movable than the left, but there is no dislocation. No fracture has taken place in any of the bones.

The point of greatest tenderness is over the left second costal cartilage. There is pain on drawing a deep breath, but this cannot be localised, nor can any crepitus or movement be felt in connection with the second costal cartilage, or with any of the other ribs.

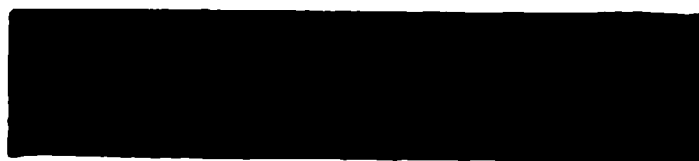
The pulse taken in the left radial was good and regular, and it was not till Mr. Cooper Forster's visit the same afternoon that the absence of pulsation in the right radial was observed. No pulsation could be felt in the brachial, axillary, or subclavian. Sensation and motion were good in this arm, and to the man himself there was nothing amiss with it.

June 20th.—On examining more carefully the site of pain, I found a sharp, crackling sound, heard with the cardiac and pulmonary movements over an area extending from the centre of the sternum to beyond the left nipple, and from the fourth rib to the margin of the ribs below. This was not heard at the base of the heart, and was loudest over the junction of the fifth rib with the sternum. This area, which included that of the normal cardiac dulness, was resonant, and the patient complained of pain over the same region. The percussion note resembled more that obtained over the stomach, and was the same up to

the fifth rib. Here also a bruit d'airain could be heard; the heart sounds could be heard distinctly to the right of the sternum, and the point of greatest pulsation was felt between the fourth and fifth ribs half an inch to the right of the sternum. There was dulness over the lower part of the sternum, extending to the right a little beyond the nipple.

21st.—Right arm and hand warmer. No pulsation to be felt in the radial, but if the brachial be compressed and suddenly liberated a slight thrill is felt in the radial, showing that blood is traversing the vessel. He is able to lie on his back without pain.

Yesterday, Dr. Mahomed took sphygmographic tracings of both radial arteries, from which it is seen that a pulsation recognisable by this instrument existed.



23rd.—He is much better. There is less pain on the left side. The dulness on the right side has diminished. A distinct double rub is heard during expiration and inspiration over the area previously noted as resonant. This rub is loudest at the junction of the fifth cartilage with the sternum, the same point at which the sharp crackling sound was most distinct. This sound suggested pericarditis, but at the base no such sound could be heard, and the area still gave the stomach resonance. The radial pulse could be felt.

26th.—The rub is not so plain. The resonance does not extend so high and the pain is less. The pulse is more distinct.

30th.—The rub has entirely disappeared; the cardiac dulness is normal; the impulse felt to the right of the sternum has also disappeared and the apex beat can be felt in the normal situation. He observed that on washing the right hand it became cold.

July 1st.—The radial pulse can be felt quite well. Got up to-day.

10th.—The hand much warmer. He was still unable to lie on the left side without pain.

The man now left the hospital. He had full use in his hand and was rapidly losing the pain in his chest.

I have recently (May, 1884) seen this man; he is in perfect health, the pulse in the right radial quite equal to the left, and there is no evidence whatever of former interference with the circulation. He says that he has pain sometimes in the left side, but there are no symptoms which suggest that at the present time there is anything wrong with the position of the stomach or heart. He is employed as a goods guard and is accustomed to lift heavy weights.

I have headed this case "injury" and not "rupture" of the subclavian artery because I am not able satisfactorily to explain the loss of pulsation and prefer to wait for further information. I may, however, call attention to the displacement of the heart, as it may have some bearing on the loss of pulsation. The pulsation in the carotid was not observed.

There is another point of great interest in this case: the thoracic injury.

Rupture of the left side of the diaphragm with hernia of the stomach or colon into the pleural cavity, causing displacement of the heart, seems to be the only explanation to the physical signs observed at the time there recorded. In this opinion I must say I could not get anyone to agree with me at the time, the general opinion being that this is a necessarily fatal injury. The case mentioned by Mr. Erichsen,¹ however, shows that recovery is possible.

CASE 5. *Rupture of brachial artery; return pulsation on fifth day.*—John Donelly, æt. 24, was admitted into Cornelius Ward under Mr. Davies-Colley November 24th, 1883. A four-wheeled waggon had passed over his left arm just before his admission. There was some swelling at the seat of injury and ecchymosis about the elbow. The pulsation in the brachial artery stopped abruptly in the middle of the arm; at this point the only swelling seemed to be due to the injury to structures external to the artery. The pulse was also absent from the radial. Some fibres of the biceps were ruptured, and there was some

¹ 'Science and Art of Surgery,' 6th ed., vol. ii, p. 561.

loss of power and anæsthesia in the index and middle fingers, indicating injury to the median nerve. A splint was applied to the arm with cotton-wool. On the 29th (fifth day) pulsation was felt in the radial for the first time. He was discharged December 8th.

On December 27th Mr. Davies-Colley examined the arm and noted that "the brachial can be felt of normal size down to the middle of the arm ; below this point only a small artery can be felt in the situation of the brachial." Pulsation was also felt in the radial and ulnar arteries, though it was weaker than on the opposite side. The superior profunda pulsated strongly, as if enlarged.

April 24th, 1884.—The hand remained somewhat stiff, but under galvanism power was returning in the index and middle fingers.

I had the opportunity of seeing this case soon after admission, when the absence of pulsation was complete.

CASE 6. *Rupture of brachial artery ; return pulsation on third day.*—Geo. Turner, æt. 11, admitted under Mr. Davies-Colley February 15th, 1884, into Accident ward, having had his right arm run over by a loaded van. The lower third of the arm, the forearm, and hand were contused and swollen, and there was an abrasion of the skin on the front of the forearm. No pulsation could be felt in the lower third of the brachial artery, nor in the vessels at the wrist. The arm was wrapped in cotton-wool, the small wound being first dressed. In the report it is definitely stated on the 17th that there was "no pulsation in radial or ulnar artery," but on the 18th—the third day—a pulse was felt in both these vessels. The median nerve had been injured at the same time, as there was some paralysis of the flexor profundus digitorum muscle. He had also sustained an injury to one of his toes, which subsequently had to be amputated. It is noted on the 27th—twelfth day—that pulsation can be felt in the whole length of the brachial artery.

CASE 7. *Rupture of temporal artery ; consecutive aneurysm.*—Littlewood, æt. 23, had been under my care at first in the hospital, and subsequently as out-patient for about a year. On May 4th, 1883, she came complaining of a lump in the left temporal region, and I made the following note :—Nine weeks before she

struck her temple against the corner of a box. She received a pretty severe blow and a bruise resulted. Two days afterwards, when the swelling caused by the injury was subsiding, the present one made its appearance. There is now a small rounded swelling, about half an inch in diameter, in the line of the anterior branch of the left temporal artery. It pulsates strongly and expansively; it can be emptied by direct compression and by occluding the temporal artery above the ear. There is no sign, as far as bruising goes, of the old injury, nor is there any pain in the swelling. At this time the only sign of active syphilis about the patient was a few mucous tubercles on the mucous membrane of the mouth.

I proposed to treat this case with direct pressure, as its small size, together with the resistance of the underlying bone, rendered it probable that this would effect a cure. Failing rapid improvement by this method, I intended to dissect out the aneurysm together with a portion of the vessel above and below the sac.

Though ligation close to the sac after laying it open is recommended in aneurysms following closely upon injuries, I should myself be disposed, when the artery is large, to treat these cases according to the method adopted in ordinary aneurysms, more especially if there was reason to suspect that atheroma existed.

The possible existence of syphilitic arteritis in this patient may, I think, be dismissed.

ON A CASE
OF
HYPERTROPHIC CIRRHOSIS,
WITH
REMARKS ON THE PATHOLOGY OF CIRRHOSIS.

By J. A. P. PRICE, B.A., M.B.

THE somewhat exceptional nature of the following case of cirrhosis of the liver is my excuse for giving in detail its clinical features, the post-mortem examination, and the microscopic appearance of the liver. I have called the case one of hypertrophic cirrhosis, although it was not accompanied by jaundice, inasmuch as I think that the term is best applied to those cases in which the morbid change results in a slow increase both in the size and weight of the liver, that organ being found after death considerably larger and heavier than in the healthy condition.

The cause of the morbid change in this case was ~~most~~ probably chronic alcoholism; at any rate, the clinical features offer no other explanation. Such a generally diffused cirrhosis is, however, rare, as a result of alcoholism, and the microscopic appearance much resembles the change seen in the cirrhotic livers of infants affected with congenital syphilis. I am not aware that anyone has recorded a similar cirrhosis in adults as undoubtedly due to syphilis. In the case of Charles C—

there was not the least evidence of either acquired or congenital syphilis. The early change in the kidneys was very slight, and in all probability due to alcoholism. The amount of ascites present was very small, the distension of the abdomen during life being due to tympanites. The slight amount of ascites and the absence of jaundice rendered the diagnosis difficult and somewhat doubtful, but the appearance of the patient and the history of many years' free indulgence in alcohol pointed to cirrhosis as the most probable cause of the hepatic disease.

CASE (reported by Mr. L. E. W. Stephens).—The patient, Charles C—, æt. 58, was admitted into Philip Ward, No. 30, under Dr. Fagge's care on December 28th, 1880, with ascites and an enlarged liver. For the last thirty-two years he had been deputy foreman at a wharf; he had never been abroad. He never had a day's illness except an occasional cough up to about twelve months ago, when he first noticed that his health was not as good as usual, that he was getting weaker, and could not stand as long as usual without feeling tired. At this time he began to lose flesh and had sinking pains in his bowels, his appetite began to fail, he became fastidious and did not feel inclined to eat without first drinking; he had no pain on swallowing.

During the last eighteen years he has been a free drinker, taking on an average five pints of beer daily with a quarter of spirits; when his appetite began to fail he drank more in order to keep himself up, but lately (during the last three months) he has taken but little alcohol and has felt worse in consequence. He kept on with his work until three months ago, when he was obliged to leave it. During the last year his urine has been very dark, quite thick at times, and on several occasions he has been unable to hold his water. He denies ever having had gonorrhœa or syphilis; he is a widower, and has never had but one son, who is now living, æt. 25, in good health. His wife never had any miscarriages. On the night before admission his feet began to swell, then his legs and abdomen—about this he is quite positive.

General appearance.—Patient is a tall man, much emaciated, his cheeks are much sunken, and his malar bones and zygomatic processes stand out. His face is covered with severe acne which has come on during the last six months; it is general over

the forehead, cheeks, nose, and lips. The patient is under the impression that it did not come out until he took some medicine.

Eyes sunken, no œdema of lids, conjunctivæ a dirty yellow, pupils equal.

Skin cold; very little subcutaneous fat.

Nails clubbed, but not typically so.

Ears normal, no tophi.

The bones, joints, and lymphatic glands seem healthy.

It was noticed that when eating his dinner he was unable to hold his fork with his left hand in a natural manner, the thumb being apparently powerless. On inquiry, he stated that as long as he could remember his left arm had always been shorter, weaker, and smaller than the right. On examination this was found to be the case. The measurement from the tip of the acromion process to the outer condyle of the humerus was found to be the same on both sides, but from the olecranon to the styloid process of the ulna the measurement on the right side was $10\frac{3}{4}$ inches, on the left $9\frac{1}{4}$ inches. Both hands are thin and wasted; the thenar and hypothenar eminences of the left hand are much wasted and flabby; the metacarpal bone of the left thumb is greatly extended so as to bring the metacarpo-phalangeal joint into a plane posterior to that of the fingers. The left hand as a whole has a clawed appearance.

Alimentary system.—The tongue is covered with a thick, dirty brown fur, fissured but not tremulous, the teeth are bad, the gums look healthy, but he says they bleed at times. His appetite is good except for meat, which he dislikes. There is great thirst at times. No nausea at present. He has never been sick in the morning, has never had hæmatemesis, has never had jaundice, but has had frequent attacks of epistaxis. The bowels as a rule are regular. The abdomen is broad and full, but no ascites is detected. The lineæ semilunares are lost, the umbilicus neither raised nor depressed. The epigastric veins are full and distinctly visible, the blood in them flows in the normal direction; on the right side the veins appear fuller than on the left. On palpation there is felt below the ribs a firm, inelastic, resistant, uniformly large, and apparently smooth swelling, bounded below by a line commencing on the right side on a level with the umbilicus, then forming a notch and passing just

above the umbilicus to descend again on the left side. Above, the swelling runs up under the ribs; it is depressed with each inspiratory movement, and is evidently the liver much enlarged. There is no tenderness over this area on palpation, but he has during the last three months complained of much pain over the right side. No bosses or nodules are to be felt; it is difficult to feel the edge of the liver. The spleen is not felt. No other tumour felt in the abdomen. On percussion the liver dulness extends as high up as the fifth space in the mammary line, and below as far as the indistinctly-felt margin of the swelling above mentioned. The splenic dulness is increased in area. The rest of the abdomen is resonant.

Respiratory system.—Voice husky, slight cough at night, little or no sputum, respiration equally thoracic and abdominal, no inspiratory or expiratory dyspnoea. The chest is very thin, the ribs are plainly visible, the sternum flat and narrow, the clavicles prominent, with depressions above and below. No suction action in the intercostal spaces. Expansion on inspiration is good and equal on both sides. The percussion note is good all over. Healthy breath sounds are heard all over the chest, with prolonged expiratory murmur behind. Tactile vocal fremitus good on both sides. Respirations 28 in the minute.

Circulatory system.—Cardiac impulse, feeble and diffused, is felt well in a line just internal to the nipple in the fifth left intercostal space. The heart sounds are normal, there are no bruits. The radial pulse is of moderate fulness, fairly strong, and compressible.

Urinary system.—No difficulty or pain on micturition; the urine is pale, acid, with a sp. gr. of 1018, no albumen, no sugar, no deposit. No scars in the groins.

January 1st.—The edge of the liver can be easily felt on a level with the anterior superior iliac spine of the left (? right) side. A hard cord can be felt running up to the umbilicus from the supra-pubic region, and on either side of the cord a hardness is felt. Dr. Fagge thought that the diagnosis lay between cirrhosis and enlargement of the liver due to malignant disease.

3rd.—Urine, sp. gr. 1022, loaded with lithates, acid, non-albuminous, a port-wine colour is produced on addition of nitric

acid. Last night he had great pain in the right hypochondriac and epigastric regions.

4th.—Circumference of abdomen at level of umbilicus thirty-one inches, midway between the sternum and umbilicus thirty-three inches. Morning temp. 99.2° . Tongue moist, centre and tip bright red. Skin hot, pungent, dry. Motions of a natural colour, no pain on defæcation.

5th.—Great pain over the abdomen on firm pressure. Morning temp. 99.2° .

6th.—There seems to be a considerable amount of fluid in the abdomen to-day. On tapping with the finger just below the umbilicus a wave is seen running up towards the thorax. The bowels have been opened by means of a soap enema. Digital examination of the rectum revealed nothing abnormal. He feels very weak and cannot walk even a few yards without feeling very tired. Morning temp. 100.4° , pulse 100. Pulsation visible in the epigastrium.

12th.—The feet this morning are œdematous, abdomen more tense, umbilical depression obliterated. He has a feeling of “something dragging on his inside.”

15th.—Circumference at umbilicus $33\frac{3}{4}$ in., at higher level $35\frac{1}{4}$ in. Yesterday for the first time he was unable to button his trousers round him. His appetite is the same, and he has an uncontrollable dislike for meat. Face much more free from acne. He does not sleep well, he can only lie on his back. He is never sick. Feet and legs œdematous. Slight epistaxis this morning.

20th.—Can button his trousers again. Severe epistaxis again.

22nd.—Circumference at umbilicus 33 in., higher level $34\frac{3}{4}$ in. Slight epistaxis.

24th.—Circumference at umbilicus $34\frac{1}{4}$ in., higher level $35\frac{1}{4}$ in.

26th.—Great pain over the epigastric region. Much flatulence. Abdomen mainly resonant and tympanitic.

27th.—Circumference at umbilicus $33\frac{1}{4}$ in., higher level $35\frac{1}{4}$ in.

28th.—Insomnia lately; was given one fifteenth of a grain of hyoscyamine in a pill, after which he slept.

31st.—Patient has become much thinner during the last three weeks.

February 2nd.—He feels very ill. Face free from acne.

4th.—Had an epileptic fit and died half an hour afterwards at 8 a.m.

Post-mortem examination (made by Dr. Fagge seven hours after death). The face was quite free from acne. On opening the abdomen a small amount of ascitic fluid was seen, the cavity was filled with distended coils of small intestine floating in the fluid.

Liver weighed 130 oz.; its surface was quite smooth and uniform. It was enormously and uniformly enlarged. On section the liver substance looked coarse and grey, the surface of the section was somewhat dry; it was extremely tough and indurated. The biliary ducts were not dilated.

Gall-bladder contained thin, light-coloured bile; its duct was patent.

Portal vein patent, walls somewhat thicker than normal and more opaque. The glands in the portal fissure were much enlarged; those close to the pancreas were red and fleshy looking. Mesenteric glands large.

Adrenals very dark coloured, almost black, otherwise healthy.

Pancreas healthy, firm.

Stomach pale, not at all reddened, showing no marked congestion of portal system.

Kidneys.—Weight 11 oz. Capsules strip well, not granular on surface; medullary part looks yellowish, as if fatty.

Spleen.—Weight 12 oz. Considerably enlarged, very fleshy, rather hard, but not extremely so.

Heart.—Weight 8½ oz., somewhat wasted, otherwise healthy.

Lungs œdematous.

Brain.—The convolutions immediately behind the fissure of Rolando appeared small and close together. Ascending parietal convolution small on both sides. No hæmorrhage, no effusion into the ventricles, no lesions whatever discovered.

Muscles of thenar and hypothenar eminences of left hand were yellow, flabby, and fibrous. Second left lumbricalis muscle was in the same condition. The spinal cord was too much damaged in removal to be of any use for microscopical examination.

Microscopical examination.—The tissues were hardened in dilute chromic acid and alcohol, and stained with a solution of hæmatoxylin.

The liver.—The chief feature is the very general distribution of a richly nucleated fibrous tissue throughout the section. This newly-formed tissue involves the lobules themselves, invading their structure and surrounding the hepatic cells, splitting the lobule up into islets of cells. The nuclei of the fibrous tissue take the staining well. Some parts of the section resemble granulation tissue more than anything else, with masses of huddled-up liver cells imbedded in it. There is no marked isolation of individual lobules or groups of lobules by strands of fibrous tissue, and the section is very unlike one of either ordinary unilobular or multilobular cirrhosis, the change being pericellular. The hepatic cells are nowhere fatty; in a few places they look fairly normal, but are perhaps a little wasted and smaller than usual. As a rule they are, as above stated, collected in groups of different shapes and sizes, and surrounded by fibrous tissue. Some of the groups are elongated strands of compressed hepatic cells; a few of the latter are in shape somewhat spindle-like. The nuclei of all stain equally well, those of the compressed cells no better and no feebler than those of the fairly normal-looking cells. The transition from the compressed hepatic cells to the strands of surrounding fibrous tissue is somewhat abrupt. The so-called new biliary ducts are well seen here and there, but they are all in the immediate neighbourhood of the portal canals. The intra-lobular plexus of veins is much distended and full of blood. Newly-formed blood-vessels are seen in the fibrous tissue.

Kidneys.—Numerous nuclei lie between the convoluted tubules, indicating an early interstitial change.

The ear muscles.—Striation almost entirely lost; an increased amount of fibrous tissue with a little fat here and there.

In order to find out as far as possible the actual facts in the pathology of cirrhosis the post-mortem reports for the years 1875—1883 inclusive were examined, and the results tabulated in all the cases of cirrhosis. A few cases may have been accidentally passed over, and several have been inserted which were clinically cardiac disease, tuberculosis, or other disease. The reason for including such cases is that they afford some

information when examining into the natural history of cirrhosis, and more especially are the surgical cases valuable for this purpose. As far as possible the actual clinical reports have in each case been examined in order to make out what was the diagnosis during life and to discover what evidence cases, admitted more than once, afforded either in favour of or against the general impression that cirrhotic livers, though enlarged in the early stage of the disease, gradually undergo a process of contraction so as to become at death much smaller than normal.

Sex.—Out of 142 cases thirty-four, or about 26 per cent., are females.

Age.—This will be referred to later on when considering the weights of cirrhotic livers.

Ætiology. Alcohol.—In seventy-two cases out of 142 there is a history of alcohol, in eight or nine cases alcoholism is probable, in a few cases it is altogether denied, and in about fifty the reports make no mention of such excess, thus leaving the matter in doubt.

Syphilis.—In only nine cases is there any evidence of syphilis, and in six of these, as might naturally be expected, it is associated with alcoholism. From the recorded appearance of the liver in these cases the cirrhosis in the greater number was probably due rather to alcohol than to syphilis. The latter is rarely the cause of a true cirrhosis, the lesion being coarser, less evenly distributed, accompanied by a more extreme lobulation of the surface of the liver than in alcoholic cirrhosis.

The number of cases in which jaundice or ascites occurred either alone or associated one with the other, the relation between the various weights of the liver and the ages of the different patients at death, the number of fatty livers, &c., are all shown in the accompanying tables.

Weights of livers in ounces.	No. of cases.	No. of fatty livers.	Jaundice only in	Ascites only in	Jaundice and ascites in	Neither jaundice nor ascites in
20—29.....	2	—	1 ¹	1 ²	—	—
30—39.....	7	1	—	6	1	—
40—49.....	20	5	4 ³	7 ⁴	4	5. None of these died of cirrhosis.
50—60.....	33	9	3 ⁵	14 ⁶	2	14. None of these died of cirrhosis.
61—69.....	15	—	2 ⁷	8 ⁸	1	4. One of these died of cirrhosis.
70—79.....	16	9	1	2	4 ⁹	9. Two of these died of cirrhosis.
80—89.....	14	5	1	3 ¹⁰	4	6. One of these died of cirrhosis.
90—99.....	11	7	1	2 ¹¹	2	6. One of these died of cirrhosis.
100—150	10	5	—	4	2	4.
150.....	2	—	1	1	—	—
Cases in which no weights are recorded	12	—	—	8	1	8.
Totals.....	142	41	14	51	21	56.

¹ A case of acute atrophy supervening on cirrhosis ; the jaundice was of about six weeks' duration, and intense.

² Had double psoas abscess and diseased vertebræ. The liver was typically cirrhotic.

³ In one of these cases jaundice was due to heart disease.

⁴ In one of these cases ascites was due to peritonitis, in a second probably to the condition of the kidney (large white). A third died from phthisis.

⁵ In two of these cases neither jaundice nor death was due to cirrhosis.

⁶ Four of these cases died from causes other than cirrhosis.

⁷ In one of these cases jaundice was probably due to heart disease.

⁸ In one of these death was due probably to causes other than cirrhosis. There was a history of syphilis, and the liver was lobulated and generally cirrhotic. As the patient was a woman alcoholism was probable though not recorded.

⁹ One of these was clinically a case of heart disease.

¹⁰ One of these was clinically a case of heart disease ; the liver was cirrhotic and not nutmeg.

¹¹ In both of these cases ascites was due to cardiac disease, which was the clinical feature of each.

Taking 50—60 oz. as the average weight of the liver it will be seen that in 29 cases only was the liver below 50 oz., and of the 29, two were children aged 14 and 16 years respectively in which the liver would normally be considerably less than 50 oz. in weight. In 68 cases the liver weighed more than 60 oz. This number, however, includes a few cases in which the enlargement may have been due to cardiac or other disease. The cases mentioned under (7), (9), (10), (11), must therefore be omitted from the list, so that we have 63 cirrhotic livers weighing over 60 oz. each.

It is necessary, however, to pursue these figures still further if we wish to find out the true weight of the cirrhotic liver. Naturally all cases in which cirrhosis was not the cause of death must be excluded from the above numbers.

Out of the 29 cases mentioned above, 21 died of cirrhosis, and two of these were children, which should also be excluded; this leaves 19 cases in which the liver weighed less than 50 oz.

Treating the 68 cases in the same way there are found to be 38 which died of cirrhosis, and in which the liver weighed upwards of 60 oz.

The cases in which the liver weighed 50—60 oz. (inclusive) are in all 33, and only 13 of these died of cirrhosis.

Thus of 70 cases ($19 + 13 + 38$) dying from cirrhosis of the liver, in 38, or over 54 per cent., the liver was increased in weight; in 13 cases, or over 18 per cent., it was of average weight; and in 19 cases, or over 27 per cent., it was below the average weight.

An examination of the figures under the heading "Neither jaundice nor ascites" will afford some information with regard to the weights of livers, which though cirrhotic gave rise to no symptoms. It will be as well to refer to these cases in detail.

Of the cases in which the liver weighed 40—49 oz., 5 had neither jaundice nor ascites.

One had a fractured tibia and delirium tremens, æt. 51; liver fatty.

One died of cerebral disease, æt. 39; liver granular.

One died suddenly, æt. 54; liver markedly cirrhotic.

One died of pyæmia, æt. 48; liver in a state of early cirrhosis.

One died of mitral disease, æt. 15½ ; liver in a state of early cirrhosis.

Liver weighing 50—60 oz. Fourteen cases without jaundice or ascites.

Seven died of phthisis, of the ages respectively of 19, 28, 35, 35, 47, 49, 53 ; in all there was an early cirrhosis, and in two the livers were fatty.

One died of opium poisoning, æt. 66 ; liver granular.

One died of extensive injuries, æt. 54 ; liver decidedly cirrhotic.

One died of cardiac disease, æt. 40 ; liver nutmeg and cirrhotic.

One died of renal disease, æt. 58 ; liver moderately cirrhotic.

One died of apoplexy, æt. 50 ; clinically acute nephritis ; advanced cirrhosis of liver.

One died of hepatic abscess, æt. 47 ; liver moderately cirrhotic.

One died of broncho-pneumonia with epithelioma of the penis, æt. 59 ; liver markedly cirrhotic.

Liver weighing 61—69 oz. Four cases without jaundice or ascites.

One died of fractured skull, æt. 60 ; liver markedly cirrhotic.

One died of senile gangrene, &c., æt. 71 ; liver cirrhotic.

One died of tuberculosis, æt. 36 ; liver much cirrhotic, contained tubercles.

One died of cirrhosis with acute mediastinal inflammation, æt. 48.

Liver weighing 70—79 oz. Nine cases without jaundice or ascites.

One died of cirrhosis (pericellular), æt. 42 ; liver very fatty.

One died of tubercular lungs and atheromatous coronary arteries, æt. 49 ; liver markedly cirrhotic.

One died of sunstroke, age not stated ; liver very cirrhotic and fatty.

One died of epithelioma of the œsophagus, æt. 66 ; diffused cirrhosis.

One died of extensive injuries, æt. 52; liver granular and fatty.

One died of cut throat, æt. 48; liver distinctly cirrhotic.

One died of tuberculosis, æt. 31; liver fatty.

One died of phthisis, æt. 36; liver granular.

One died of empyema and pneumothorax, æt. 36; liver granular.

Liver weighing 80—89 oz. Six cases without either jaundice or ascites.

One died of thoracic aneurism, æt. 36; liver cirrhotic.

Two died of fractured skull, æt. 34 and 45; liver cirrhotic, fatty.

One died of cardiac disease, æt. 43; liver cirrhotic.

One died of pulmonary embolism, age not stated; liver fatty.

One died of cirrhosis and cystitis, æt. 43.

Liver weighing 90—99 oz. Six cases.

One died of cirrhosis, with pneumonia and dilated heart, æt. 32; liver fatty.

One died of acute pleuro-pneumonia, æt. 45; liver fatty.

One died after lithotomy, age not stated; liver intensely cirrhotic, very fat.

One died of dysentery, æt. 21; liver extremely cirrhotic.

One died of compound fracture of tibia and fibula, æt. 57; liver very fatty and cirrhotic.

One died of cerebral hæmorrhage, æt. 35; liver fatty and cirrhotic.

Liver weighing 100—150 oz. Four cases.

One died of pyæmia, age not stated; liver very fatty and much cirrhotic.

One died of fibroid phthisis, æt. 24; liver fatty.

One died of fractured femur and pneumonia, æt. 35; liver fatty.

One died after amputation of leg, æt. 48; liver cirrhotic and very fatty.

Grouping together from the above list those cases, eleven in all, in which death was due to an accident, and the patient, therefore, until the time of injury presumably in sufficiently

good health to pursue his daily work, the average age at death is found to be exactly fifty years, the average weight of the liver exactly 76 oz., and in seven out of the eleven that organ was fatty. These facts show conclusively that, whatever be the ultimate condition of the cirrhotic liver, it is at a comparatively early stage and throughout the greater duration of the disease larger and heavier than in its healthy state, and very fatty.

Do these enlarged cirrhotic livers contract and become smaller and lighter than the normal viscus? Dr. Bright¹ was uncertain whether this change took place, but states that if it did, then the result was a hobnail liver.

With the hope of throwing some light on this question I examined the reports of those patients who were admitted more than once with cirrhosis, and who ultimately died in the hospital. As a rule, patients with cirrhosis came into the hospital to die, so that from this source I did not gain as much information as I at first expected. This perhaps is not to be wondered at when we consider how small a proportion of the whole is formed by the contracted cirrhotic livers (*vide* above statistics), and when we remember that the liver may be considerably cirrhotic without giving rise to symptoms.

The four following cases are taken, two from our clinical reports and two from the 'Transactions of the Pathological Society:'

1. Girl, æt. 9, admitted into Miriam Ward under Dr. Moxon July, 1869, with ascites and enlargement of liver and spleen. Extent of liver dulness in right mammary line six inches, extending from the fourth rib to two and a half inches below the margins of the ribs.

On admission in August, 1879, the liver could not be felt below the ribs, and at death it weighed 35 oz., its surface being hobnail.

2. A woman admitted under Dr. Pavy in 1874 with ascites and considerable enlargement of the liver (the measurements were not stated) was readmitted in 1877 with ascites and died. Post mortem: the liver weighed 53 oz. and was extremely indurated and cirrhotic.

3. In the 'Transactions of the Pathological Society'² Dr.

¹ 'Abdominal Tumours,' New Syd. Soc., 1861, p. 280.

² Vol. xxxi, p. 120.

Frederick Taylor records a case of cirrhosis in a child. The patient was under observation for three years, during which time the liver, which was at first enlarged, slowly contracted.

4. In another volume¹ of the same 'Transactions' will be found a case by Dr. Griffiths in which, when first observed, the liver was found to be greatly enlarged, and at death, a year afterwards (at the age of ten), it was very small, weighing only 15 oz.

It would have been more satisfactory had the above all been adults, but as it is they go to show that the cirrhotic liver, though at first large, does contract and occasionally even to a small size. That this is not the rule, but rather the exception, is proved by the facts previously stated, that in only 27 per cent. are cirrhotic livers below the average weight.²

An examination of the third column in the table on p. 303, showing the number of cases in which the liver was found to be fatty, proves that it is the heavier and larger livers which are so affected and to the greatest extent. Taking this fact together with the large proportion of fatty livers noticed in those cases where death was accidental, one must conclude that when cirrhotic livers diminish in size the decrease is in part due to loss of fat. This agrees with the observations of Dr. Wilks and Dr. Moxon, as stated in their work on Pathological Anatomy.³ In a past volume⁴ of these reports the late Dr. Hilton Fagge remarks as follows :

"On casting up the ages of persons in whose bodies cirrhosis of the liver was discovered, without there having been marked symptoms during life, I find that the average weight was higher by about five years than that of those persons who died of the effects of the disease. If this fact can be relied upon, and if it should be confirmed by a wider experience, I think it would prove that cirrhosis is not always a progressive disease, but rather that, after having reached a certain degree of development, it often remains stationary." This statement is borne out when we compare the average age of those persons who, having cirrhotised

¹ Vol. xxvii, p. 187.

² I do not wish to imply that livers heavy at death may not have been some time previously still heavier.

³ P. 147.

⁴ Vol. xx, ser. 3, p. 193.

livers, died from accidents with that of those who died from cirrhosis. The figures are 50 as against 45·01, very nearly a difference of five years in favour of those who did not die of cirrhosis.

The large cirrhotic livers represent a more acute form of the disease than do those of average or less than average weight ; death occurring at an earlier age in patients with the former than in those with the latter.

The average age at death is shown in the following table :

A. Liver 60 ounces or less.

(a) With jaundice and ascites . . .	57·0 years.
(b) With jaundice alone	46·6 „
(c) With ascites alone	44·42 „

B. Liver above 60 ounces.

(a) With jaundice and ascites . . .	48·0 years.
(b) With jaundice alone	42·6 „
(c) With ascites alone	40·1 „

It will be seen from the above figures that those patients with ascites alone died at an earlier age than those with jaundice. This is in all probability due to the fact that several of these cases had tuberculosis, and that the latter, though not the actual cause of death, may have accelerated the end.

Ascites, as is well known, is a frequent symptom in cirrhosis. Thus, out of seventy-two cases, ascites was present in fifty-eight, or about 80 per cent. In several instances it was associated with jaundice. It would also seem that the heavier the liver at death the less frequently is it accompanied by ascites.

Jaundice is a far less frequent symptom ; out of seventy-two cases it was present in thirty, or about 41 per cent., in ten of these unassociated with ascites. Amongst these cases of jaundice those with the heavier livers were to those with the lighter ones in the proportion of about three to two. Where death was preceded by coma, jaundice was almost invariably present. In calculating the above percentages it must be mentioned that those cases are excluded in which jaundice was due

to causes other than cirrhosis, as well as those in which, although the liver was cirrhotic, death was probably due to some concomitant disease, such as disease of the heart, kidneys, or lungs.

The spleen and kidneys in cirrhosis.—The spleen is in the greater number of cases enlarged, and very frequently indurated or firm. Enlargement more especially characterises the cases with heavy livers. In many cases where the spleen was found after death enlarged, the increase in size had not been made out during life.

The association of renal disease with cirrhosis has been frequently noticed. Out of the 142 cases tabulated the kidneys were either granular or had wasted cortices in twenty-five, or about 18 per cent. If, however, those cases only be taken in which death was due to cirrhosis the figures stand thus:

Liver 60 ounces or less.

No. of cases.	Granular kidney, &c.
34	3 = 8·8 per cent.

Liver above 60 ounces.

No. of cases.	Granular kidney, &c.
38	9 = 26·4 per cent.

The heavier livers then show a far higher percentage of granular kidneys. This is in accordance with the statement that the kidney is more frequently found to be granular in hypertrophic than in atrophic cirrhosis.

It has been suggested by some pathologists that cirrhosis may occasionally be due to a fibroid induration consequent on the deposit of tubercle in the liver. If this ever occurs it must be rare, as otherwise there would frequently be found caseous masses scattered throughout the liver tissue.

Out of 140 cases the cirrhotic liver was found to contain tubercles in three cases only; in one of these there was a history of alcoholism, in the remaining two nothing was said on this point. The following table includes the above three cases, and indicates in how many tubercle was found in other organs.

	No. of cases.	Tubercle in.
Liver below 60 oz. or less .	62	16
„ above 60 „ . . .	68	10
Weight of liver not stated .	12	4

Thus out of 142 cases tubercle was present in the form of phthisis, tubercular peritonitis, &c., in thirty cases. Out of about seventy-five cases dying from cirrhosis, tubercle was found in eleven.

It is possible that the presence of tubercle elsewhere than in the liver may produce such a state of ill health or even such a condition of the liver that the stimulus of alcohol would act more readily in these than in other individuals. Examining the fatty livers of a few children with extensive tubercular disease of viscera other than the liver, I have found considerable nuclear proliferation in the portal canals and very evident biliary canaliculi, resembling somewhat an early cirrhosis; it is conceivable that such a condition might go on to cirrhosis. Whether the nuclear proliferation was connected rather with the great amount of fatty change within the hepatic cells than with the general condition of the patient I know not.

I will conclude these remarks with a brief allusion to the minute anatomy of the cirrhotic liver.

To allude once more to the minute anatomy of the liver in the case of Charles C—, I have been fortunate enough to be able to compare it with a preparation made by Mr. C. J. Symonds, from the cirrhotic liver of an infant, aged three months, with congenital syphilis. The resemblance between the two preparations is very striking. In both there is a large amount of fibrous tissue within the hepatic lobules compressing and isolating groups of liver cells; the difference between the two is, that the syphilitic cirrhosis seems to be of a less active nature, as indicated by the fewer nuclei existing in the new-formed fibrous tissue, and that in the liver of Charles C— there is on the whole less destruction of the hepatic parenchyma.

In referring to the account of the microscopical anatomy of the cirrhotic liver, I will confine myself to a brief consideration of the parts played by the hepatic cells themselves, and the nature of the so-called new-formed bile ducts.

The changes undergone by the hepatic cells are either active or passive, and in some cases they co-exist.

The passive changes, such as atrophy and fatty degeneration, are too well known to need further mention.

The active changes are of far greater interest. In one and the same section it may be often noticed that whilst the central cells of the hepatic lobule are disintegrating those of the periphery are undergoing active changes, as indicated by the proliferation of their nuclei, followed by an increase in the number of cells themselves. This change has been well described by Dr. Hamilton.¹ In some places more than two nuclei can be seen within one cell, in others, a collection of nuclei seemingly derived from the hepatic cells. The further change undergone by these proliferated cells is doubtful. Dr. Hamilton believes that the greater amount of the fibrous tissue in the cirrhotic tissue owes its origin to the liver-cells themselves. Such a change is, I think, extremely doubtful, and after the examination of several specimens of cirrhosis of different kinds I have not been able to make out any connection between the hepatic cells and the fibrous tissue beyond that of contiguity. It is extremely difficult to say for certain that in any case the fibrous tissue in the neighbourhood of the existing hepatic cells owes its origin to these. I have observed cells, exactly similar to those figured by Dr. Hamilton, in the fibrous tissue which had been formed from the perivascular connective tissue within the hepatic lobule.

Further, against Dr. Hamilton's theory is the fact that in no other gland, as far as I am aware, has fibrous tissue been observed to arise from the secreting structure.

The statement that the hepatic cells arise from the mesoblast, a structure which plays so great a part in the later formation of fibrous tissue, is not in accordance with the more recent descriptions² of the development of the liver. According to Balfour, the hepatic cells are derived from that embryonic layer which gives rise to the mucous membrane and the glandular structures of the alimentary tract from the pharynx to the lower end of the rectum. To show of how little avail are arguments deduced from embryology either for or against the question at issue, it need only be pointed out that whilst those

¹ 'Journ. Anat. and Physiol.,' vol. xiv, p. 192.

² Quain's 'Anat.,' 9th ed., vol. ii; "Embryology of the Chick," Foster and Balfour.

pathologists who are inclined to believe in the origin of fibrous tissue from the hepatic cells, appeal to the mesoblastic origin of the latter in support of their view, others instance the hypoblastic origin of hepatic cells as explanatory of the formation of new bile ducts therefrom.

It is possible that this cell proliferation may play another and more important part in the ultimate fate of the cirrhotic liver. There are a certain number of cases of cancer of the liver which seem to show that this disease, in the absence of any primary source elsewhere, may be secondary to cirrhosis. It is quite conceivable, though difficult of proof, that this malignant change is due to an extreme activity in the hepatic cells, the slighter extent of which is so frequently observed in cirrhosis.¹

The presence of duct-like structures has been explained in a variety of ways. They have been regarded as newly-formed bile ducts, as merely the nuclei of hepatic cells arranged in double rows, and finally, as the proliferated nuclei of the perivascular connective tissue within the lobules. The latter certainly do give rise to the appearance of ducts, but careful examination shows their relation to the vessels and not to the hepatic cells; the nuclei, too, appear to be elongated and not round.

As far as I have been able to make out from the preparations which I have examined, there appear to be two distinct conditions, described under the term "new bile ducts." Firstly, there are very evident biliary ducts, always seen at the periphery of the lobules and in the portal canals; these are undoubtedly bile ducts, as is seen by their relation to the branches of the portal vein and hepatic artery, and their easily recognised appearance and apparently increased number is due to the proliferation of their lining epithelium on the one hand (the ready staining of the nuclei indicates an active change going on at the time of the death of the patient), and, on the other, to the fact that the contracted liver tissue brings a greater number into the field at the same time, just as is observed in the case of the Malpighian glomeruli of the kidney in chronic interstitial nephritis. I have observed this condition in affections of the liver other than cirrhosis, i.e. in the cicatrices in syphilitic livers, in the

¹ 'Atlas of Path.,' Fasc. iv; 'Summary of Diseases of the Liver,' Goodhart, New Syd. Soc., p. 87.

neighbourhood of inflammatory foci secondary to abscess in the liver, in the neighbourhood of the injury in ruptured liver, most marked of all in the liver of lymphadenoma, where the secreting tissue of the organ had almost entirely disappeared. When present in cirrhosis this appearance is by no means invariably accompanied by jaundice.

The second condition, to which I allude, is the presence of duct-like structures with large tracts of fibro-nucleated tissue. These too are found in cases of cirrhosis either with or without jaundice. Their origin and nature are difficult to make out; they consist of definitely nucleated cubical cells arranged around a central lumen; they stain well but not as readily as the ducts mentioned above. In some of my specimens I fancy I can trace a gradual transition between isolated groups of liver-cells and these duct-like structures, the latter ultimately disappearing where the new-formed fibrous tissue is most dense. The association in some cases of these duct-like structures with jaundice and enlarged liver led Charcot to describe hypertrophic cirrhosis as having its origin in the bile ducts from obstruction of the latter, as being in fact a biliary cirrhosis. Dr. Saundby,¹ however, has shown that these structures are not necessarily associated with jaundice, being present in some cases of cirrhosis without jaundice, and absent in others when jaundice was present.

Whether cirrhosis can arise from irritation or obstruction of the bile ducts is uncertain. Thinking that the condition of the liver in sheep with flukes might throw some light on the question, I examined some sections prepared by my friend Mr. George Turner, one of which he has kindly drawn for me (Fig. 3). I found that there was a considerable increase in the amount of fibrous tissue of Glisson's capsule with a large number of nuclei which were invading the periphery of the lobules. Here and there were also seen duct-like formations. The presence of flukes in the bile ducts does not necessarily imply obstruction of the latter, so that the above changes may have been the result of irritation of the ducts from the presence of the distoma. I think, then, that we have here a definite biliary cirrhosis, one in which the irritation must have started from the bile ducts themselves. I have unfortunately not had time to

¹ 'Path. Trans.,' vol. xxx.

examine as thoroughly as I could wish other specimens of fluked livers.

It is somewhat unfortunate that the term hypertrophic cirrhosis has been applied and appropriated to those cases of enlarged livers which are accompanied by jaundice, since it leaves no place for those cases of enormously enlarged livers without jaundice and with no great amount of ascites. It would be better to describe all cases under the general term cirrhosis, recognising the fact that there are clinically a few well-marked cases in which the liver is enlarged, in which jaundice is present, and in which death is very frequently preceded by coma.

I will conclude with a brief enumeration of the main points of this paper :

1. The cirrhotic liver is, as a rule, larger and heavier than normal.
2. The large cirrhotic liver is more fatty than the small.
3. Shrinking of the primarily enlarged liver does take place, and is due in part to loss of fat.
4. The large cirrhotic livers represent a more acute form of the disease than do the small. This is indicated by the fact that death occurs at an earlier age in cases with the former than in those with the latter.
5. A considerable degree of cirrhosis may exist without causing symptoms, and the disease, after reaching a certain stage, may remain stationary.
6. The spleen is, as a rule, enlarged, more especially in those cases where the liver is also increased in size.
7. Granular kidney is more frequently associated with the large than with the small livers.
8. Death may be preceded by coma whether the liver be enlarged or not, and is in the greater number of cases associated with jaundice.

I am greatly indebted to Dr. Goodhart for the opportunities afforded me of examining numerous sections of cirrhotic livers, and to Mr. George Turner for the drawings which accompany this paper.

In the following table the weights of the viscera are given in ounces.

An Abstract of the Post-mortem Examinations of 142 cases in which the Liver was Cirrhotic.

No.	Jaundice: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease, &c.	Condition at death, &c.
1	—	74 Lymph on capsule; tubercles underneath; organs very fatty and cirrhotic throughout	Tubercular	Tubercular	Thick cerebral arteries, caseous mesenteric glands, pneumonia, tubercular peritonitis	Delirious.
2	None	73 Pale yellow and fatty, and very firm and tough; liver-cells universally fatty; a diffused pericellular cirrhosis; several so-called new bile ducts seen amidst the large amount of fibrous tissue which exists everywhere	8; large and soft	8; red and congested	Slight tubercular disease of uterus, Fallopian tubes, and ovaries	Comatose.
3	Slight for months	48 Hobnail surface; very hard and tough	Healthy	16; very congested	—	Died quietly.
4	None	70 Surface very nodular and pale; organ very tough and fatty, markedly cirrhotic	24; caseating nodules in substance; white patches on capsule	17½; tubercles	Miliary tubercles in lungs; atheroma of aorta and coronary arteries	Exhausted.
5	None	69 Much capsulitis. Right lobe much shrunken, dense, hard, and much smaller than the left. Strands of fibrous tissue traversed the organ in various directions. Extreme cirrhosis. Surface hobnail. Microscopically like cirrhosis, but appears to be cancerous; no cylindrical cell formation, but	Large and soft	17; calculus in left; otherwise healthy	Chronic and acute peritonitis, pleuritic effusion and compressed lung	Collapsed.

6	M., 68	—	—	For 4 months	Yes	98	larger and different from a cir- rhotic formation. Probably cir- rhosis becoming cancerous Surface slightly irregular; early cirrhotic changes; hypertrophic cirrhosis	20; much enlarged and soft; capsule white and thick	14½; con- gested, with shrunken cortex	Emphysema and œdema of lungs	—
7	F., 57	—	Yes	Yes	For 3 weeks; had pre- vious attacks	82	Surface granular; on section very firm and tough. Multi- and uni- lobular cirrhosis. Minute bile ducts well seen at periphery of wasted lobule. Hepatic cells con- tained oil drops, several broken up into a granular <i>débris</i> Cirrhosed and fatty	15; large; some recent inflamma- tion of capsule	9½; granular; cysts	Edema of larynx and lung; gout	Delirious.
8	M., 49	—	Yes	—	—	56	5; white patches on capsule	5; white patches on capsule	14; tubercles	Phthisis, tuber- cular testes; ad- herent pericar- dium	Asthenia from phthisis.
9	F., 40	—	Yes	Yes	Faint tinge	98	Hard and tough, surface smooth; cirrhosed with much fat	8; normal consistence	8½; granular	Miliary tubercles in lungs; dilated R. ventricle; tu- bercular ulcera- tion of intestine	Comatose after taking a quantity of gin.
10	F., 58	—	—	For 3 months	Very alight	45	Surface granular; very tough	5	11; surface slightly granular	Double pleuritic effusion	Comatose.
11	M., 35	—	—	—	—	56	Cirrhosed, especially in the region of the left and spigelian lobes	8½; rather large and a little soft	12½; healthy	Phthisis; old and recent pleurisy	Died of phthisis.
12	M., 52	—	—	Consider- able	Intense	78	Fatty, nutmeg, and cirrhosed	18; yellow infarcts	12; yellow infarcts	Atheroma of aorta, dilated and hyper- trophied heart; acute pneumonia; gout	Comatose.

Ascites: its duration.	Jaundice: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease.	Condition at death, &c.
For 9 due to peri- tonitis	—	86 Capsulitis; cirrhotic and lobular; very tough	35	12	Old syphilitic dis- ease of skull; em- pyema, and sup- purative perito- nitis; gumma be- tween spleen and liver No primary source of cancer found	—
For 5 months	Has had jaundice	34 Shrunken; surface only slightly granular. On section pale, firm, and dense; evidently a great amount of interlobular fibrous tissue	7; some thickening of capsule	10; normal	Broncho-pneu- motia; tubercular peritonitis	Comatose.
—	—	59 Slightly granular, but not tough	4½	9½; granular	Opium poisoning; thickened cere- bral vessels	Had been drinking heavily be- fore he took the opium.
For 4 months	None	60 Enlarged, fatty and cirrhotic. A large amount of nucleated fibrous tissue throughout the liver; very evident duct-like structures in the fibrous tissue. Hepatic lobules wasted and cells of sinusoids, in addition to new proliferation	26; tuber- cular	10	Hydrothorax; tu- bercles in left pleura; tuber- cular peritonitis	—

	F.,	—	—	For months	Decided		Edge rounded, capsule thickened; extensive cirrhosis. An ordinary amount of fibrous tissue surrounding individual lobules. Evident minute bile ducts in the fibrous tissue at periphery of lobules	19; capsule thick and opaque. Tissue scarcely harder than natural	13; surface granular	Pneumonia	Comatose; much wasted.
18	—	—	—	—	—	40					
19	M., 52	Yes	Denied	Slight for a month	None	130	Surface smooth, section nearly so, extreme induration. Pericellular cirrhosis. (Case of Charles C—, reported above)	12; indurated	11; large, firm and blurred	Wasting of left thenar and second left lumbricalis muscles	—
20	F., 35	Yes	—	—	—	55½	Very hard, and appears cirrhotic	12; opaque patches on capsule	8; cortex wasted	Phthisis; tubercular ulceration of larynx and of intestine	—
21	M., 40	—	—	Due to peritonitis	—	40	Mottled, purple and white in small patches; smooth and very hard	3; capsule thick and opaque	10; healthy	Chronic phthisis; peritoneum thick and opaque	—
22	M., 59	Probable	Yes	None	—	54	Markedly hobnail; excessive cirrhosis in all parts. Uni- and multilobular cirrhosis. Large tracts of fibrous tissue with numerous nuclei. Very evident biliary canaliculi at periphery of lobules. No duct-like structures elsewhere. Liver cells have a homogeneous appearance, margins indistinct, nuclei distinct and stain well	—	10; healthy	Epithelioma of penis; bronchopneumonia	—
23	F., 45	Yes	—	—	Yes	161	Adhesions on surface. Shape distorted, edge thick and rounded. Surface yellow. Tough. Fatty and very tough. Nuclei of hepatic cells proliferating. Bile ducts very evident at periphery of lobules, which are being invaded by the fibrous tissue. Venules dilated	—	19; not unhealthy looking Cystic	Had delirium tremens	—
24	F., 28	—	—	—	—	55		—		Phthisis; tubercular ulceration of intestine; wasted brain	—

No.	Sex and age.	Alcohol.	Syphilis.	Ascites: its duration.	Jaundice: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease, &c.	Condition at death, &c.
25	M., 36	Yes	Yes	—	—	83 Surface uneven from wasting of gland-tissue beneath, a number of depressions over the surface	16½; pale, capsule thick	13; cortex swollen and blurred, acute nephritis	Aneurism of thoracic aorta; broncho-pneumonia, ulceration of trachea, ulceration of rectum	—
26	M., —	—	—	—	—	78 Very cirrhotic and fatty	7	11; congested	Brought in dead from sunstroke	—
27	M., 42	Yes	—	Considerable for 5 weeks	Slight	108 Not easily lacerable, despite post-mortem changes; apparently fibrous throughout	12	14; pale, fatty, striation faint	Pleurisy, vomica in left lung; had dysentery and ague	—
28	M., 48	Yes	—	—	—	— A good size; much granulated all over, looked to be in an advanced state of cirrhosis, fatty. Capsule not thick	—	—	Thickened membranes; death due to acute alcoholism	Comatose.
29	M., 32	—	—	Yes	—	76 Highly cirrhotic, granular, and full of fibrous tissue. Portal vein at portal fissure thick and obstructed by adherent thrombus	48; tough	12	Pariform lymphoma	—
30	M., 18	—	—	Yes	—	— Enlarged, irregularly lobulated, a cluster of bosses here and there, with comparatively smooth and	5½; firm and thick cartilage-like patch on surface	—	Ulceration of colon, induration of mesentery and subperitoneal fat; fibroid phthisis and ulceration of larynx	—
31	M., 40	—	—	Set in rapidly near end of life	None	88 Yellow nodules surrounded by a great amount of grayish matter diffused throughout the hepatic tissue. No caseation. Micro-	14; no tubercles	—	Phthisis; tubercular disease of peritoneum	—

32	M., 28	Yes	Yes	—	None	—	Indurated, fatty and cirrhotic	—	—	Phthisis; ulceration of larynx	—
33	M., 36	—	Yes	—	Yes	52	Granular surface, tough and fibrous on section	4	13; indurated	Mitral stenosis; pulmonary embolism, ante-mortem coagula in right auricle	—
34	M., —	—	—	—	—	108	Fatty and much cirrhotic	13; soft	14½; coarse	Amputation of leg; pyæmia	—
35	M., 49	—	—	—	—	—	Large and heavy; surface irregular tending to hobnail; some lobules congested, grey ramifying material in others. Liver-cells in places slightly fatty and atrophied, fibrous tissue increased in amount	Pulpy	—	Laryngitis, ulceration of larynx; fungating endocarditis	—
36	M., 24	—	Denied	For one month	—	103	Irregular, capsule thick; great increase in fibrous tissue; lobules not well defined	21; capsule thickened; increased fibrous tissue	13½; firm, congested, infarcts	Adherent to pericardium; general dropsy; pyrexia	—
37	F., 58	—	Yes	4½ mos.	—	92	Very hard and firm, becoming distinctly hobnail on surface	26; large and firm	13; congested	Dilated heart	Clinically mitral regurgitation.
38	M., 47	—	Yes	—	—	54	Surface and section studded with numerous small yellow tubercles. Granular and very tough from cirrhotic changes	8; healthy	9; indurated	Phthisis, thickened pleura	Died exhausted from phthisis.
39	M., 61	—	Yes	—	Slight for more than a month	43	Capsule thick, surface very granular; a good specimen of ordinary cirrhosis. For drawing, vide 'Syd. Soc. Atlas of Path.,' fig. 4, pl. xviii	9½; very soft	13½; large, parenchymatous nephritis	Chronic peritonitis; old apoplexy in left optic thalamus	Died suddenly from cardiac condition.

No.	Sex and age.	Alcohol.	Syphilis.	Ascites: its duration.	Jaundice its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant diseases, &c.	Condition at death, &c.
40	M., 53	Yes	—	Yes	—	62 Capsule thick, surface very granular; mottled on section, some parts depressed, others raised. Excess of fibrous tissue. For drawing, vide 'Syd. Soc. Atlas of Path.,' fig. 1, pl. xvii	8½; capsule thick	10½; much congested	Tubercular peritonitis; thick mitrals; double hydrothorax	Sank gradually, much wasted.
41	M., 38	—	—	—	Slight	60 Fatty and cirrhotic	—	19; yellow and fatty	Amputation of thigh, double pneumonia	—
42	M., 46	0	—	—	For 6 weeks	76 Extensive and diffused cirrhosis of upper and anterior parts of liver; the hinder part of the right lobe was softer than the rest of the organ. Liver-cells full of oil drops	7½; very pulpy	10; capsules adherent granular cortex	—	—
43	F., 34	Yes	Yes	—	—	84 Granular and fatty; cicatrices on surfaces with stellate fibrous patches and gummata beneath	15½; large and soft	11½; granular	Fractured skull; contused brain	—
44	M., 34	Yes	—	For 9 weeks	—	60 Extremely cirrhotic, portal vein thickened and dilated	9; indurated	9; healthy	—	—
45	F., 50	Yes	—	For a year more or less	—	67 Capsule greatly thickened and alveolated on surface; universally cirrhotic	11; capsule thickened in patches	Granular	Chronic peritonitis	—
46	M., 66	—	—	—	—	77 Surface minutely granular; diffused cirrhosis	—	—	Epithelioma of oesophagus and soft palate	Died from effects of growth.
47	M., 23	0	Yes	Off and on for years	—	66 Capsule thickened, granular on section, mottled, no gummata, no tubercles. Lobules separated by broad bands of richly nucleated fibrous tissue and in some places masses of infiltrating cells	8; indurated	18; congested	Chronic tuberculosis	—

48	F., 53	—	—	—	78	Fatty, tough, surface granular	—	—	—	Extensive injuries, pericarditis	—
49	F., 55	Yes	—	—	54	Cirrhotic, adherent by lymph to neighbouring organs	Healthy	Healthy	—	—	—
50	M., 56	Yes	—	—	41	Extensive cirrhosis, greater part of organ converted into a white greyish mass. Unilobular cirrhosis, fibrous tissue spreading between hepatic cells and compressing them into parallel rows, resembling bile ducts. Hepatic cells much wasted but in places proliferating, vacuolation taking place in the nuclei to start with. Iary canaliculi at obules. In the midst of masses of fibrous tissue are structures resembling bile ducts	26; indurated, opaque patches on capsule	10; healthy	Erysipelas	—	—
51	M., 48	Yes	—	Extreme	65	A good specimen of hobnail liver; an extreme amount of fibrous tissue in some parts, in other parts more corresponding to multilobular cirrhosis of Charcot	9; soft and pulpy	18½; healthy	Sugar in urine	Cometose.	—
52	M., 48	Yes	—	—	77	Granular; distinctly cirrhotic	4; soft	18; congested Granular	Cut throat, pneumonia, pleurisy	—	—
53	M., 42	Yes	Yes	—	—	Marbled with firm white and grey stripes; slightly granular	—	—	Fibroid testes, hypertrophied heart, had had lead colic	Clinically granular kidneys.	—
54	M., 26	0	—	—	—	Cirrhotic, contained tubercle	—	—	Tubercular meningitis, tubercle in lungs, scrofulous disease of cervical, mediastinal, and abdominal glands	—	—
55	M., 45	Yes	—	For 1½ month	54	Extremely cirrhotic	7; capsule very opaque	11; healthy	Bronchitis	—	—

No.	Sex and age.	Alcohol.	Syphilis.	Ascites : its duration.	Jaundice : its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease, &c.	Condition at death, &c.
56	M., 54	—	—	—	—	57 Decidedly cirrhotic, bile ducts large	3; shrivelled	12; rather coarse	Fractured ribs, tibia, and clavicle. Old pleurisy, with destruction of lung	—
57	M., 40	Yes	—	—	—	58 Nutmeg, rather extensively granular, due to a primary cirrhosis, not to a cardiac condition	8; firm	14; congested, wasted, granular, surfaces puckered	Bronchitis, emphysema, dilated bronchial tubes, bovine heart	—
58	M., 50	Yes	—	Abdominal distension for 5—6 months	9 years before, but not during recent illness	46 A good specimen of hobnail liver, without any of its toughness. A tawny colour on section. Liver-cells healthy, except where they contained much granular fat. A slight excess of fibrous tissue	15; pale, capsule irregularly thickened	15; pale but healthy looking	Suppurative peritonitis after paracentesis; gummatous testes; adherent pericardium	—
59	M., 60	—	—	—	—	61 Marked cirrhosis	10½	9; wasted	Fractured skull	—
60	M., 26	—	—	—	Yes, P due to heart	69 Very granular section, not on surface, very tough	25½; old embolism	16; very congested	Adherent pericardium hypertrophied and dilated heart. Mitral disease	Clinically cardiac.
61	F., 32	Bloated appearance	—	None	—	92 Fatty and tough; surface smooth. General portal cirrhosis in the cellular stage. Very few biliary canaliculi seen	18; rather soft	15; congested, inflamed	Dilated heart, oedema of lungs; pneumonia	—
62	M., 25	Yes	—	Very severe ascites	None	102 Fatty granular on surface, much on section. Intersected by many small nodules	47; soft and pulpy	15; healthy	Suppurative oedema of lobules	—

Case	Sex	Age	0	0	For 9 weeks	—	56	Adherent to diaphragm by old lymph; nodular on surface, tough and yellowish on section. Evidently cirrhotic	10; slightly lardaceous	11; surface irregular, not healthy	Acute peritonitis	Delicious.
64	M.	24	Yes	—	—	—	100	Orange yellow, very fatty and very tough	6; healthy, firm	14; congested	Nodular fibroid phthisis	—
65	M.	58	—	—	—	—	58	A little granular on surface, moderately cirrhotic, pale and fatty	9; healthy	Much wasted and diseased	Stricture, cystitis, pyelitis, perineal fistula	—
66	F.	44	Yes	—	Yes	None	50	Very granular and tough; grey and fibrous on section, with yellow points of liver substance between bands of fibrous tissue	4; capsulitis	18; subacute nephritis	—	Died quietly.
67	F.	30	—	—	Loculated	—	98	Irregular surface: on section fatty between atrophic liver tissue. Much distended.	9; rather soft	13; indurated	Adherent pericardium, hypertrophied and dilated heart	Clinically cardiac.
68	M.	45	—	0	Yes	—	50	Some cirrhotic change Surface very granular; on section fatty portions in between cirrhotic parts	16; soft	9	Phthisis both apices; tubercular ulceration of larynx; extreme colitis	—
69	M.	35	—	—	—	—	100	Light yellow, fatty, very firm; much fibrous tissue	—	13; seemingly healthy	Fractured femur, pleurisy, pneumonia	—
70	F.	51	A beer drinker	—	Considerable, ? due to heart	—	80	Cirrhotic, tough, congested, not netmeg	17	16; surface irregular	Hypertrophied and dilated heart; adherent pericardium. Thickened mitral	Clinically cardiac.
71	M.	48	—	—	—	—	88	Capsule thick, adherent to diaphragm. Tough, cirrhotic, no cardiac condition	27; firm, capsule thick and adherent to surrounding parts	—	Fibroid disease of heart; old adhesions of pericardium with calcareous changes	Died suddenly, probably from heart.

No.	Sex and age.	Alcohol.	Syphilia.	Ascites: its duration.	Jaundice: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease, &c.	Condition at death, &c.
72	M., 14	—	—	For 23 days	—	22 Typical example of cirrhosis; surface light yellow and granular	6; probably lardaceous	6½; no reaction with iodine	Diseased lumbar vertebrae, double psoas abscess	—
73	M., 51	Yes	—	—	—	48 Very tough and fatty	8½	14; surface granular, capsules adherent	Fractured tibia	Delirium tremens.
74	F., 19	—	—	Considerable for a month	—	35 Contracted; extensive cirrhosis; surface hobnail	30; firm	10½	Tubercles in lungs	—
75	F., 32	—	—	For 9 weeks	—	— Small; extensive cirrhosis of the diffused granular form	—	Capsules adherent	Phthisis	Low muttering delirium, wasted.
76	M., —	Probable	—	—	—	88 Fatty, very hard and tough	4; healthy	13; granular on surface, mottled	Pulmonary embolism. Hypertrophied left ventricle	Uræmia, comatose.
77	M., 31	—	—	—	—	79 Some cirrhosis; irregularly fatty	8½; tubercles	18; tubercles	General tuberculosis	—
78	F., 41	Yes	—	For 5 weeks	Slight	77 Surface nearly smooth; hard, mottled on section. Fatty. Liver cells well formed; fibrous tissue with granules which showed no nuclei when treated with acetic acid	11; firm	13; congested, inflamed	Fatty heart	Comatose.
79	M., 71	Yes	—	Yes	—	50 Very cirrhotic and granular	6	15; tubular nephritis	Calcification of aortic valves, causing stenosis	Clinically bronchitis and cardiac disease.
80	M., 60	Yes	—	Yes, for 9 weeks	—	42 Moderate cirrhosis; capsule thick	Capsule thick	9; appeared healthy	Chronic peritonitis	Emaciated; unconscious.

81	F., 58	Yes	—	At least 7 weeks	Slight	37	Small; capsule thickened and stripping with difficulty, smooth; advanced cirrhosis	8; decomposed	13; capsules adherent	—	—
82	F., 45	Yes	Yes	None	19 days, deep	90	Surface minutely granular; heavy and tough, hardly any healthy tissue left. An extreme amount of fibrous tissue; liver-cells very fatty and atrophied. Many nuclei within fibrous tissue, also numerous duct-like structures	13; rather firm	12½; healthy looking	Collapsed lungs	Comatose.
83	M., 53	Yes	—	Yes	—	57	Cirrhotic and fatty	13; soft	6; granular and wasted	Chronic peritonitis, tubercular ulceration of intestine; phthisis, gout	Died suddenly, wasted.
84	M., 45	Yes	—	For 4 months	Yes	68	Cirrhotic, tough, hobnail; dense strands of fibrous tissue throughout	9; flabby, anemic	11; healthy, except for a few cysts	Edema of lungs, pleurisy	—
85	M., 45	Yes	—	None	None	90	Fatty and rather granular	7; soft	13; healthy	Acute pleuro-pneumonia	—
86	M., 25	Yes	?	—	Deep for 5 days	85	Flocculent capsule, scarred in places; organ showed cirrhosis and a mottling like a nutmeg liver. Also number of bright yellow patches. Septa all thick, if liver-cells still fatty and disintegrated of hyaline fibroid material in portal fissure (? canal), but no cells	38; capsule thick and cartilaginous	21; large, swollen and coarse	Double pneumonia; fibroid testes; ichthyosis of tongue; pericarditis	—
87	F., 30	Yes	—	About 11 weeks	Some time previously, not when in hospital	50	Indurated, much fat in liver-cells, jaundiced, no gummata. An ordinary cirrhosis. Where fibrous tissue is most abundant there are collections of cells resembling bile ducts	7; firm and dark colour	11; healthy	Double pneumonia	Wasted.
88	M., 53	Denied	—	—	—	53	Finely granular, diffused early cirrhosis	6; healthy	14; healthy	Phthisis, ulceration of epiglottis	Died of phthisis.

No.	Sex and age.	Alcohol.	Syphilis.	Ascites: its duration.	Jaundice: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease, &c.	Condition at death, &c.
89	F., 39	Yes	—	—	—	47 Much indurated, slightly granular on surface and on section	Healthy	Healthy	? Atrophy of brain	Comatose.
90	M., 71	—	—	—	—	68 Granular on surface; cirrhotic	—	—	Chronic arteritis, senile gangrene, cedema of lungs and larynx	—
91	— 43	?	?	None	—	88 Fissured into large lobules in all directions; adhesions to diaphragm	26; thickening of capsule	Healthy	Cystitis; fibroid testis	Emaciated.
92	M., 71	Yes	—	For 12½ weeks	For 8 weeks; disappeared before death	50 Nodular on surface; smooth and homogeneous on section. Ordinary cirrhosis with remarkable hypertrophy of some hepatic cells, which were double the normal size, and contained each two nuclei	9; capsule very thick and opaque indurated substance	Healthy	Chronic and acute peritonitis	—
93	M., 36	—	—	—	—	70 Cirrhotic, granular, but soft	12; large, soft	16; quite healthy	Phthisis; emphysema of lungs	Brought in dead; had been a soldier.
94	M., 55	—	—	For 4 weeks	—	41 Surface minutely granular; on section mottled and fatty; diffused general cirrhosis	8; capsule thick, tubercles	13; healthy	Old phthisis; tubercular peritonitis and pleurisy; ulceration of intestine	Died of phthisis.
95	M., 52	Yes	—	—	Deep for some weeks	67 Advanced cirrhosis; very much indurated; granular on section	6; small and firm	Large, soft, healthy	Bronchitis; emphysema; pleurisy	Dellious.
96	M., 60	Always taken	—	—	—	58 Advanced cirrhosis	11½; soft	11½; mottled interstitial nephritis	Apoplexy	Clinically acute nephritis.

97	M., 47	Yes	—	For 6 weeks	For 6 weeks	—	Extreme cirrhosis; covered over with an opaque separable mem- brane; surface and section smooth Intensely cirrhotic and very fat	27; capsule opaque, soft —	Large, appeared healthy Coarse and flabby	Chronic peritonitis	Delirious.
98	M., —	—	—	—	—	95				Lithotomy for cal- culus vesicæ; ul- ceration of sto- mach	—
99	F., 43	Yes	—	For 3 months	—	53	Extremely indurated and cirrhotic. Thrombosis at junction of splenic and superior mesenteric veins, but not occluding vessels. A typical cirrhosis	10; not indurated	Uneven surface, capsules adherent, interstitial change	Acute peritonitis after tapping	Wasted.
100	M., 48	Yes	—	None	Yes	47	Surface smooth; organ very tough and cirrhotic throughout	3; firm	Healthy	Large heart; pleu- ritic effusion; pneumonia; gout	Comatose; wasted.
101	M., 47	Yes	—	—	—	60	Six small abscesses in liver; mode- rate cirrhosis	5	9	Ulceration of cæ- cum and rectum	Died of hepatic abscess.
102	M., 57	—	—	—	—	—	Markedly cirrhotic	Healthy	Healthy	Phlegmonous ery- sipelas of thigh	—
103	M., 15	Pro- bable	—	Yes	—	32	Surface hobnail; a remarkable specimen of simple cirrhosis	22; capsule rather thick	12; indu- rated, hard, somewhat congested	Peritonitis; ostitis of right femur	Comatose.
104	M., 45	Yes	—	—	—	85	Markedly cirrhotic, hobnail, pale, and fatty	7; rather soft	13½; con- gested	Fractured skull; contused brain	—
105	F., 48	Yes	—	For at least 3 weeks	—	32	Surface a little irregular; whole organ of a bright ochre-yellow colour like a case of acute yellow atrophy, but firmer. Ducts and arteries normal. Portal vein plugged entirely in the main trunk and its larger branches. Liver structure microscopically fairly normal, except very fatty; fibrous tissue in portal canal in great excess	16; capsule thick, tough and bloodless	9; healthy	Chronic peritonitis; œdematous lungs; adherent pericar- dium	—

No.	Sex and age.	Alcohol.	Gyphalia.	Ascites: its duration.	Janasides: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant diseases, &c.	Condition at death, &c.
106	F., 42	Yes	—	—	—	— Very tough, smooth on surface and section. An advanced cirrhosis, lobules everywhere separated by a substance, and attended by the nuclei, in and with the lei of recent leucocytes. In some places shrunken masses of hepatic cells enclosed in new fibrous material	Moderately firm	Healthy	Atrophy of brain	—
107	F., 56	—	—	For at least 6 weeks	For at least 6 weeks	58 Tough, capsule a little thick, surface nodulated; hepatic ducts much dilated; gall-stones in gall-bladder. A large amount of fibrous tissue with here and there destruction of liver-cells	36; tough, hypertrophied, not lardaceous	10; healthy	Dilated heart	—
108	M., 48	Yes	—	None	None	108 Extremely granular surface, and circumscribed all through; very fatty	9; very soft and fever-like	16; surfaces not good	Primary amputation of leg	Delirious.
109	M., 36	Yes	—	For months	—	102 Large, roughened, and cirrhotic	32; large, hard	Right 4; granular. Left 6; mottled	Anæmia	—
110	M., 46	—	—	For at least 3 weeks	Deep for 3 weeks	104 Hobnail all over; tawny, large, almost all fibrous tissue, with but little thickening or adhesion of capsule. Large tracts of fibrous tissue around the lobules, with biliary canaliculi well marked at the periphery. Embedded in fibrous tissue are groups of cells resembling biliary ducts	16; rather soft	13; healthy	Old endocarditis, with thickening of aortic and mitral valves	Wasted, muttering delirium.

111	M., 54	—	—	None	—	41	Very granular; on section strands of fibrous tissue between, which are islets of normal-looking hepatic lobules	6½	9; congested	Edema of lungs; Died rather suddenly.
112	F., 50	—	—	—	Slight, due to heart disease	49	Capsule opaque; tissue granular and cirrhotic	Capsule opaque	10½; puckered surface	Chronic disease of aortic and tricuspid valves
113	M., 38	Yes	—	For at least 4 months	—	53	Capsule rather thick; organ small, on section granular, tougher than normal. Increase of fibrous tissue in portal canals, but not infiltrating lobules to any extent	31½; pale, flabby, capsule a little thick	18½; red, smooth, inflamed	Pleuritic effusion; acute and chronic peritonitis
114	M., 36	—	—	—	—	65	Cirrhotic to a considerable extent; a few scattered tubercles	Healthy	14; numerous tubercles	Caseous tubercle of medulla oblongata; tubercular ulceration of intestines; tubercles in lungs
115	M., 21	—	0	—	—	96	Looked average size; surface finely granular from extreme cirrhosis	25; tough	12; healthy	Dysentery; early lardaceous disease of intestine; adherent pericardium; chronic peritonitis
116	F., 48	—	—	—	—	44	In a condition of early cirrhosis	5; somewhat wasted	Cortex wasted	Carcinoma of nose, cheek, skull; pyæmia.
117	M., 41	Yes	Yes	Yes	—	47	Surface somewhat granular, with some thickening of capsule; granular; no section; no fibrous scars; not very tough	9; firm, patches of thickening on capsule	26; large, white	Tubercular phthisis; ulceration of intestines; fibroid testis
118	M., 63	?	—	For 3 months	—	46	Capsule adherent; section granular; well-marked cirrhosis	9; very soft	19; congested	Bad vessels
119	M., 41	Mode-rate	Denied	For 3 months	—	67	Covered all over with tough lymph, which could be peeled off; tough and granular on section	8; quite healthy	18; healthy, horse-shoe shaped	General peritonitis

No.	Sex and age.	Alcohol.	Syphilis.	Abscess: its duration.	Jauddies: its duration.	26	15; soft	13; swollen	Concomitant diseases, &c.	Condition at death, &c.
120	M., 22	Yes	—	—	Intense for 5—6 weeks	<p>Capsule opaque, but not thick; surface nodulated. On section broad bands of fibrous tissue surrounding ochre-yellow liver substance which were distinct; other tracts reddish and quite unlike liver tissue. Microscopically the ochre-yellow portions were seen to be formed of huddled-up liver-cells, showing by their arrangement that large tracts of liver tissue had contracted down. Liver-cells were in process of atrophy, deeply pigmented and granular, with intervening bright orange pigment grains</p>				Comatose.
121	M., 36	Yes	—	—	—	<p>Granular and slightly tough</p>	10; abscess	12; granular	Dilated bronchial tubes; pneumonia thorax; empyema	—
122	F., 16½	—	—	—	—	<p>Indurated, fatty, granular on section; commencing cirrhosis</p>	Small and hard	14; epithelial nephritis	Mitral stenosis, acute peritonitis	—
123	F., 46	—	—	—	Deep	<p>Considerably cirrhotic</p>	12; firm and hard	Healthy	—	Comatose.
124	F., 54	—	Yes	Yes	—	<p>Capsule very thick, surface cut up into several large lobules; general cirrhosis, in some places grey patches of fibrous tissue with ill-defined borders</p>	12; capsulitis	9; healthy	Epithelioma of pudenda, peritonitis, osteitis of tibia, atheroma of aorta	—
125	M., 43	Yes	—	For 6 months	Deep	<p>Capsule but slightly thickened; tough on section; whole organ</p>	7; firm	11; healthy	—	Comatose, thin.

126	M., 43	Yes	—	Moderate	—	112	permeated by a grey semi-transparent fibrous material Surface slightly irregular; exceedingly tough, large amount of fibrous tissue in portal canals; fatty	11½; rather soft	14½; acute nephritis	Thickened brain membranes, pneumonia	Rambling, comatose.
127	F., 46	—	—	For 5—6 months Yes	Slight	71	Very large and hard; very cirrhotic	18; firm	10½; granular	—	Delirious.
128	M., 55	Yes	—	Yes	—	42	Surface very granular	2½; very soft	14; healthy	—	Comatose.
129	M., 18	—	—	Yes	—	32½	Small and puckered, no thickening of capsules; markedly cirrhotic	5½; healthy	9½; pyramids pale	—	Uræmic.
130	M., 19	Yes	—	—	—	51	Capsule opaque; early cirrhosis	6½; healthy	11½; healthy	Pneumonic phthisis, tubercular disease of intestine	—
131	M., 57	—	—	—	—	90	Very fatty and cirrhotic	3½	9½; granular	Compound fracture of right tibia and fibula	—
132	M., 39	—	—	Yes, due to peritonitis	—	—	Thickened capsule; markedly cirrhotic	Healthy	healthy	Phthisis; ulceration of larynx and of intestine; tubercular peritonitis	—
133	M., 48	Yes	—	None	None	67	Granular on section; advanced cirrhosis	6; normal	13; good	Pneumonia and double pleurisy	Delirious.
134	M., 43	Yes	—	Yes	—	56	Small; on section nodules of fat-like material. Excess of fibrous tissue in portal canals. In the lobules several hepatic cells were hyaline-looking and devoid of pigment	5½; flaccid	12½; looked healthy	Phthisis, ulceration of larynx and large intestine	—
135	F., —	—	—	Yes	Very slight	81	An extreme degree of cirrhosis	Soft, but healthy	Soft, but healthy	—	—
136	M., 34	Probable	No history	Yes	—	66	Cirrhotic. Fibrous tissue much increased	Much enlarged	9; recent emboli	Morbus cordis, vegetations on aortic valves	—

No.	Sex and age.	Alcohol.	Gyphilia.	Ascites: its duration.	Jundice: its duration.	Liver. Weight, naked-eye and microscopic appearance.	Spleen. Weight, &c.	Kidneys. Weight, &c.	Concomitant disease, &c.	Condition at death, &c.
137	M., 35	Probable	—	—	—	91 Large, fatty; increase of 1	5; very soft	9; healthy	Cerebral hemorrhage	—
138	M., 45	Yes	No history	For 3½ months	Yes	48 Surface smooth, capsule thickened and adherent; great increase of fibrous tissue; bands seem to run in from capsule	14; firm	11½; cortex somewhat wasted	Recent peritonitis	Died quietly.
139	P., 61	Yes	—	For 4 weeks	For 4 weeks	67 Firm, tough, fatty; capsule thickened	9	11	Fatty degeneration of heart	—
140	M., 41	—	No	For some weeks	For some weeks	82 Surface rough and nodular; thickened connective tissue in portal canals; tough	15½; large and soft	15; appeared healthy	—	Comatose.
141	M., 54	Yes	No	Considerable for 2½ months	None	38 Very small, finely granular on section, surface intersected by fibrous bands. A large amount of fibrous tissue invaded the intimate structure of the lobules, in places there was scarcely any liver structure left	6½; firm	Healthy	Chronic phthisis, acute pleurisy with effusion; ulceration of intestines	Exhausted.
142	M., 40	Yes	—	Yes	—	63 Very granular on section	8; capsule thick and opaque	9; mixed interstitial and catarrhal nephritis	Apoplexy of pons	Semi-comatose.

DESCRIPTION OF PLATE,

Illustrating Dr. Price's case of Hypertrophic Cirrhosis.

FIG. 1 represents a section of the liver of Charles C., under a $\frac{1}{2}$ inch obj.

FIG. 2. The same, under Zeiss D obj.

- a.* Portal canal, with much fibro-nucleated tissue.
- b.* Fibro-nucleated tissue within lobule and compressing liver-cells.
- c.* Rows of atrophied compressed liver-cells.
- d.* Small vessels.
- e.* Minute bile duct in transverse section.
- f.* Space probably due to method of preparation.

FIG. 3. Section of sheep's liver. *Vide* p. 314.

- a.* Hepatic cells, with space between columnar rows.
- b.* Bile duct.
- c.* Vein in portal canal.
- d.* Nuclei spreading into lobule from portal canal.



Fig 1

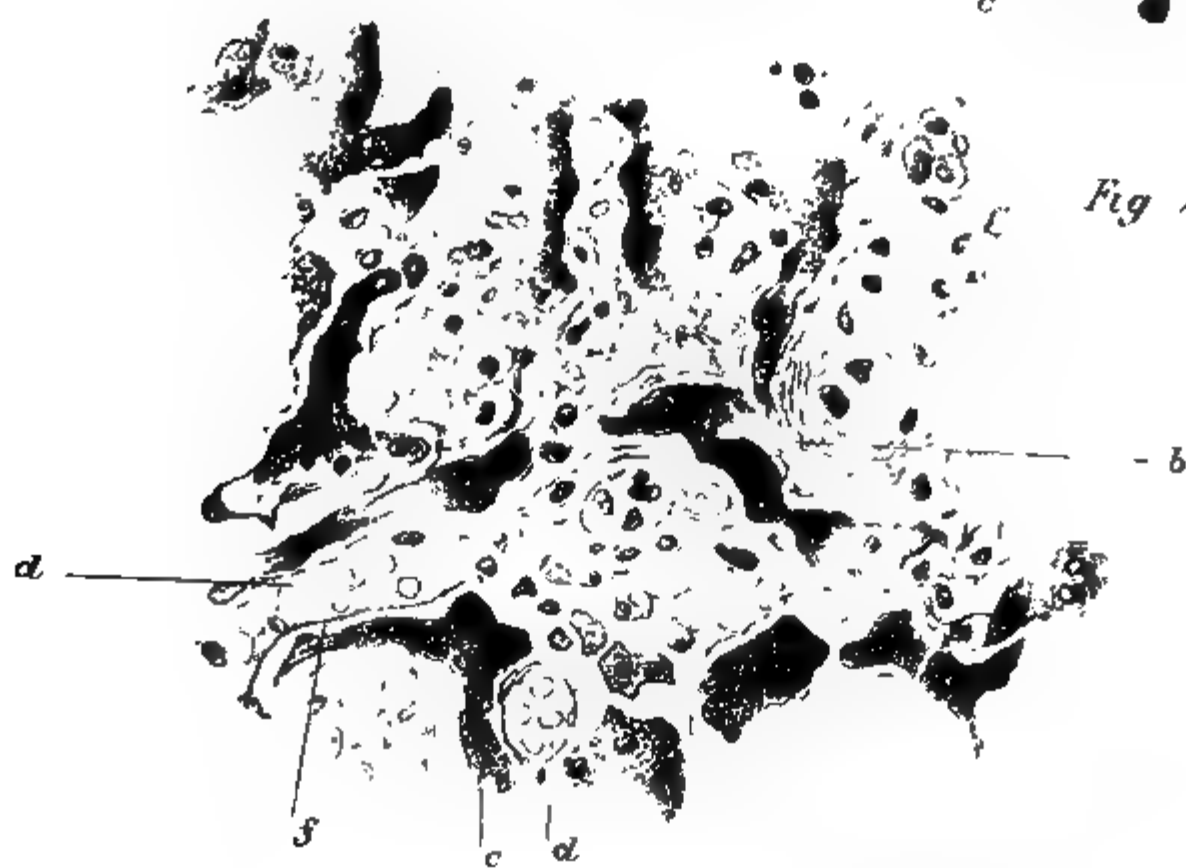
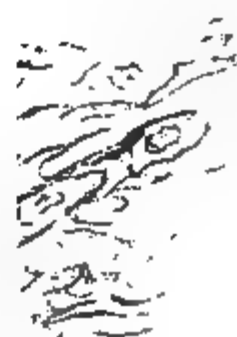


Fig 2



Fig 3.



A CASE
OF
ENLARGED, SO-CALLED "HYPERTROPHIC,"
CIRRHOTIC LIVER;

WITH
OBSERVATIONS ON THE CONCURRENCE OF FEVER
WITH CIRRHOSIS.

By R. E. CARRINGTON, M.D.

THE patient, Elizabeth H—, æt. 42, was admitted into Guy's Hospital on June 6th, 1883, under the care of Dr. Wilks, to whom I am indebted for permission to publish the case, which has been reported by Mr. H. E. C. Cooper.

She was a married woman, and had had one miscarriage, but no children.

The family history was remarkably good.

She had no occupation further than her domestic duties.

She had been in the habit of drinking ale in moderate quantity, and never took spirits. There was no history of ague.

The menstrual flow had always been abundant, especially since her marriage, but it had now ceased for over a year.

History of present illness.—She enjoyed fair health up to four months prior to her admission. She then began to feel weak, to vomit constantly after her meals, to suffer from profuse diarrhoea, and at night from burning sensations in the soles of her feet, and to sweat a great deal. She also began to lose flesh

rapidly. The vomiting gradually became aggravated in severity, it took place chiefly in the early morning when she sat up in bed, and without any previous ingestion of food; but she was also liable to it throughout the day after her meals. There was no pain. The diarrhoea also increased to the extent of from five to seven evacuations daily. The dejecta were liquid and offensive, but she had never noticed them to contain blood.

Her legs and feet began to swell an hour or so after rising in the morning. She had been attending the out-patient department for two weeks prior to her admission, but without any relief to her symptoms.

Condition on admission.—She looked anæmic, with a slightly yellow tint of skin, but the conjunctivæ were pale and white. The general surface was moist. No enlarged glands were to be felt. She looked worn and ill, and maintained the recumbent posture, lying low in the bed.

The tongue was moist, and covered with a slight white fur.

The liver dulness, in the mammary line, reached from the sixth rib to two inches below the costal margin. The anterior edge could be felt at the lower limit. The right rectus abdominis muscle was somewhat rigid, and the hepatic region was tender. The splenic dulness was not increased. Her appetite was bad. There had been no vomiting since admission. The bowels had acted and the fæces were streaked with blood. Examination per rectum caused little or no pain, and nothing abnormal could be detected.

No abnormal physical signs could be made out in the respiratory or circulatory systems. There was a slight cough but no expectoration. She breathed twenty-nine times to the minute. The pulse was small and weak, the number 110.

The urine was somewhat scanty and high coloured, sp. gr. 1010. It contained neither albumen nor sugar.

Her mental faculties were good, but she was somewhat irritable.

The temperature chart will be subjoined in a tabular form at the end of the report.

She was ordered milk and farinaceous diet, and Pulv. Doveri, gr. x, at night.

June 12th.—The tongue and fauces were dry, and there was

a slight white fur on the former. Marked sordes on lips and teeth. The bowels acted on an average once in twenty-four hours. The liver was increased in size, its lower border being distinctly felt three inches below the margin of the thorax, in the mammary line. The hepatic region was markedly tender. At noon of the preceding day, after eating some rice pudding, the patient was seized with pain in the epigastrium and left hypochondrium, and this had persisted since. Her cough was a good deal worse, but there was no expectoration. The breathing at the left apex in front was very much louder than at the corresponding region of the opposite side, and a sibilant rhonchus was occasionally audible. At the inferior angle of the left scapula, first with inspiration and subsequently with expiration, a few clicking sounds could be heard. There was no dulness, nor any other abnormal sign at any part of the chest.

On June 13th the Dover's powder was discontinued, and she was given three grains of quinine every four hours.

14th.—The louder breathing at the left apex was still fully appreciable, and percussion resonance was impaired at the same spot; but nothing further could be detected in any part of the chest. The liver was in the same condition as noted on June 12th, but by no means so tender. The surface was smooth, and the organ felt hard.

16th.—The tongue was moist and covered with white-brown fur; sordes were present on the teeth and lips. The appetite was bad. She complained of gnawing epigastric pain, chiefly after taking anything to eat or drink. She was constipated. No blood was passed with the fæces. The whole abdomen was rather tender. The liver maintained the same size as in the former note. The skin was of an earthy yellow look, but no conjunctival jaundice was present. Respiration was still louder at the left than at the right apex, but there were no moist sounds. Pulse 112. Respirations 32. Perspiration was profuse, and she shivered a good deal in the course of the day. The urine was of sp. gr. 1025. It contained an abundant deposit of urates, but neither albumen nor bile. She was ordered Pulv. Doveri, gr. v, quartis horis.

17th.—She was ordered brandy, \mathfrak{z} iv, and Pulv. Doveri, gr. v, at night.

21st.—She remained in the same condition generally, as in the preceding note, but there was on this day marked hepatic tenderness, and percussion of the region caused persistent pain for some time. The following report of the condition of the urine refers to the four preceding days:—"High coloured; abundant deposit of urates; free from albumen, blood, and bile."

23rd.—Patient was lying low down in the bed. There was a sallow tinge of the skin, with congested venules on the face. The conjunctivæ were white and pearly. The lips anæmic. The tongue red and dry. The liver reached to the level of the umbilicus. It was not markedly tender. Its surface was smooth. No peritoneal rub could be heard over it. Nothing abnormal could be discovered in the respiratory or circulatory systems. She shivered after being washed in the morning, and a feeling of "cold water running down her back" persisted all day. She sweated copiously at night. She was quite clear intellectually.

25th.—She had now some diarrhœa, but all other conditions remained the same.

It would be tedious and useless to detail the daily notes that were made of her condition, for she varied but little from time to time. I will therefore content myself by mentioning any noteworthy circumstances that arose.

On June 28th fine crepitations could be heard in the left axilla, but there was no dulness. She also developed a systolic apex murmur.

On June 30th she had an attack of vomiting, and the bowels acted three or four times during the day. She was ordered—

℞ Ammon. Chlor., gr. x ;
Ex Aq. Flor. Aurantii, ʒj, t. d. s.
Pil. Sapon. co., gr. iij, quartis horis.

On July 2nd the patient looked very ill. She had been sick throughout the preceding night and this continued all day. The systolic murmur and moist sounds in the chest had completely disappeared. The pulse was feeble, 124, and the respirations numbered 32. The hepatic dulness remained the same. The shivering and feeling of chilliness had passed off. The Pil. Saponis co. was omitted and five grains of quinine were given
ur hours.

3rd.—She was much weaker and lay in a semicomatose state. There was great tenderness over the liver. Diarrhœa was very considerable. Pulse 120, resp. 32. She was ordered—

Inject. Morph., gr. $\frac{1}{4}$, statim ;
Quinise Sulph., gr. v, sextis horis ;
Enema Opii.

4th.—The patient was semicomatose. The tongue was dry, brown, glazed, and fissured. Diarrhœa was still profuse. The fæces were light in colour, and did not contain blood. The liver was very tender. Vomiting had ceased, but the diarrhœa persisted. The following was the condition of the urine : sp. gr. 1020, no albumen, blood nor bile, no excess of phosphates nor chlorides ; a large amount of indican was present, which was converted into indigo-blue by heating with nitric acid.

The patient gradually became completely comatose, and died at 4.10 p.m.

She was twenty-nine days in the hospital, so that her illness lasted about five months.

The record of the temperature is as follows :

	A.M.		P.M.		A.M.		P.M.
June 7.	101.8°	...	100.2°	11.	99.7°	...	99.8°
8.	101°	...	99°	12.	101.8°	...	
9.	100.2°	...	100°	13.	99.6°	...	101.6°
10.	102.1°	...	103°	13.	99.6°	...	101.6°

After this it was taken every three hours.

June 14.	A.M. 9.	...	99°	9.	...	98.4°
	12.	...	100.6°	12.	...	98.8°
	P.M. 3.	...	104°	16.	P.M. 3.	100.2°
	6.	...	103°	6.	...	102°
	9.	...	102.4°	9.	...	103.4°
	12.	...	100°	12.	...	101.4°
15.	A.M. 3.	...	98.4°	17.	A.M. 3.	100.2°
	6.	...	98.4°	6.	...	99.4°
	9.	...	98°	9.	...	99.2°
	12.	...	99.8°	12.	...	98.4°
	P.M. 3.	...	102°	P.M. 3.	...	98.4°
	6.	...	100.2°	6.	...	99.6°
	9.	...	102.8°	9.	...	102.2°
	12.	...	101.6°	12.	...	101°
16.	A.M. 3.	...	100.2°	18.	A.M. 3.	100.6°
	6.	...	99°	6.	...	99.4°

A Case of Enlarged, so-called

A.M.	9.	...	98.4°				12.	...	100°	
	12.	...	99.2°			21.	A.M.	3.	...	99.4°
P.M.	3.	...	100.4°					6.	...	99.4°
	6.	...	101.6°					9.	...	98.4°
	9.	...	103°					12.	...	99.8°
	12.	...	100.4°				P.M.	3.	...	100.6°
19.	A.M.	3.	...	99.8°				6.	...	102°
	6.	...	100.2°					9.	...	100°
	9.	...	99°					12.	...	99°
	12.	...	98°			22.	A.M.	3.	...	101.2°
	P.M.	3.	...	101°				6.	...	100°
	6.	...	103°					9.	...	98.8°
	9.	...	101.4°					12.	...	98.4°
	12.	...	100°				P.M.	3.	...	102°
20.	A.M.	3.	...	100.8°				102.4°
	6.	...	100.2°			23.	A.M.	3.	...	102.2°
	9.	...	98°					6.	...	101.2°
	P.M.	3.	...	100°				9.	...	98.2°
	6.	...	100.4°				P.M.	9.	...	101°
	9.	...	102.2°					12.	...	101.8°

Subsequently the temperature was taken every six hours.

24.	A.M. 6.	...	98°				12.	...	103°
	P.M. 6.	...	100.8°				P.M. 6.	...	102.6°
	12.	...	101.8°			30.	A.M. 12.	...	101°
25.	A.M. 6.	...	100°				6.	...	100.6°
	12.	...	102°				12.	...	100.8°
	P.M. 6.	...	103.6°			July 1.	A.M. 6.	...	100.8°
	12.	...	102.4°				12.	...	99.2°
26.	A.M. 6.	...	98.6°				P.M. 6.	...	98°
	12.	...	100.4°				12.	...	98.8°
	P.M. 6.	...	102.2°			2.	A.M. 6.	...	99.2°
	12.	...	99°				12.	...	102.4°
27.	A.M. 6.	...	101.4°				P.M. 6.	...	104°
	12.	...	103.4°				12.	...	103°
	P.M. 6.	...	100.4°			3.	A.M. 6.	...	102.4°
	12.	...	100.2°				12.	...	100.4°
28.	A.M. 6.	...	99.6°				P.M. 6.	...	104°
	P.M. 6.	...	98.6°				12.	...	103.4°
	12.	...	99.8°			4.	A.M. 6.	...	103.2°
29.	A.M. 6.	...	102.6°				12.	...	103°

The autopsy was made by myself twenty-three hours after death. The body was very much wasted.

The skin presented a slight yellowish tinge, but the conjunctivæ were white.

There was no disease of any of the cranial bones, nor of the external ear.

The cerebral membranes and arteries were all healthy and the brain itself was quite normal.

The lungs were emphysematous in the upper, and comparatively airless in the lower lobes.

The larynx, trachea, and bronchi were all healthy.

The pericardium contained a small quantity of clear yellow fluid. There were no subserous hæmorrhages.

The heart weighed eleven ounces. The muscular tissue was soft, pale yellow, and flabby. There were some small atheromatous patches on the mitral and tricuspid valves, otherwise the organ was healthy.

There was local peritonitis about the liver in the form of recent adhesions; the organ was, however, readily detached from its seat. There were two patches of old lymph in Douglas's pouch.

The stomach was extremely congested at the cardiac end, and here there were submucous extravasations.

The mucous membrane of the small intestine was pale and resembled wash-leather in appearance. The large intestine was injected. No part of the alimentary tract exhibited the lardaceous reaction.

The liver weighed seventy-two ounces. It cut very firmly and its structure could scarcely be broken down by pressure with the finger. In colour it was of a pale yellow. The ducts and veins were throughout quite healthy. The organ was not lardaceous. The gall-bladder contained about one ounce of yellow fluid bile.

Microscopical examination of the organ exhibited a marked degree of cirrhotic change. This not only involved the circumference of the lobules but extended inwards between the hepatic cells. There was a considerable degree of newly-formed fibrous tissue, but a very marked feature was the enormous amount of nuclear proliferation. The great majority of the liver-cells were distended with fat, and very few healthy cells remained.

It appeared that there was no duct proliferation, for the capillary ducts seen in a section, if as numerous as in a normal liver, were certainly not more so.

The pancreas and suprarenal capsules were healthy.

The spleen weighed eight ounces, and was somewhat enlarged and softened.

The kidneys presented nothing noteworthy.

The ovaries contained minute cysts, about the size of peas, two or three in each. They also presented some small caseous nodules. The Fallopian tubes were enlarged to the size of the little finger, and caseous throughout; the disease extended from the mucous surface outwards, but the peritoneal covering was quite healthy.

The fundus uteri contained a small intramural fibroid the size of a small walnut, and some degree of caseous change similar to that found in the tubes, extending from the mucous surfaces outwards.

The mucous membrane of the uterus was caseous and shreddy.

The shoulders, elbows, wrists, hips, knees, and ankles were examined and found quite healthy.

It is important to remark that the changes found in the uterus, ovaries and tubes were taken altogether, slight and quite quiescent; and from these facts, therefore, it *appeared in the highest degree improbable that they could have given rise to any of the clinical symptoms of the case.*

I have published the preceding case chiefly as an addendum to my paper, in the last volume of the 'Reports,' on "Multiple Small Abscesses of the Liver," and I venture to think that it is of considerable interest from a clinical point of view.

The patient came into Guy's with four months' history of illness, during which time she had been suffering from progressive weakness, emaciation, vomiting, and diarrhoea.

Whilst under observation the salient points were, that she looked worn and extremely ill. She lay low down in the bed, and was much wasted. She suffered from profuse sweats, which were often nocturnal. She had no distinct rigors, but she often suffered from shivering and a feeling of coldness. The tongue was for the most part dry, red, and glazed, and sordes were present on the teeth and gums. The liver was obviously enlarged and often excessively tender. She had no jaundice, but presented an earthy, sallow look. Her urine, though repeatedly examined, never contained biliary pigment, and the fæces were bile-stained; diarrhoea and vomiting were prominent symptoms. Her pulse and respirations were always quickened, and the range of

temperature was febrile throughout, often as high as 102° or 103°, and occasionally 104°, though sometimes falling to the normal.

These symptoms were observed from June 6th to July 4th, but it is not possible of course to say how long they preceded her admission, except that the vomiting, diarrhoea, emaciation, and loss of strength had been present for four months.

The results of an analysis of the symptoms of the cases of "multiple small abscesses of the liver" which I published last year, seemed to point to the following conclusions as to the clinical features of this affection :

1. The duration was very variable.
2. The patient looked extremely ill, and the illness appeared to be abdominal.
3. Wasting was a prominent result.
4. Sweating was neither prominent nor constant.
- 5, 6, 7. Abdominal distension, pain, and tenderness, generally in the hepatic region, were almost universally present, and the liver was enlarged to a greater or less degree in every case.
8. A dry brown tongue and sordes on the teeth and gums were pretty constant.
9. Vomiting was present in half the cases, and the liability to diarrhoea or constipation was about equal.
10. Rigors were more often absent than present.
11. Jaundice was only present in about one third of the cases, and only in two was it intense.
12. The temperature was always irregularly febrile, and the pulse and respirations were quickened.
13. Finally, the causation was in most of the cases due to some suppuration or ulceration in the area of origin of the portal vein.

I ventured to suggest that clinically "a typical case would probably be one presenting all the symptoms just detailed," together with a history of "some primary cause of hepatic suppuration," but that "such a case would be rare, and that there would be all gradations down to complete obscurity."

I was not, however, prepared for the fact that cirrhosis of the liver might completely simulate these cases of multiple suppuration, as is shown by the instance I have recorded ; and as I think a perusal either of the record, or of the short abstract

I have already well repeatedly shown. The resemblance may go so far as to suggest an acute form of possible intestinal obstruction.

It is very well known that I said if the symptoms I have mentioned are taken up by me I can be objected that they may all be symptoms of cirrhosis of the liver. But it was the concomitance of all these other more or less striking in this case. The history of the patient was of one suffering from typhoid fever. I very much doubt whether it would be generally admitted that the symptoms with fever, shivering, sweating, and a rapid rising liver are the clinical aspects of cirrhosis as I have said.

The resemblance of the present case will no doubt have the effect of increasing the uncertainty which already surrounds the diagnosis of cases of multiple hepatic suppuration and indeed of cirrhosis of the liver: but I venture to quote what I said last year in my presentation for doing so, "that to differentiate the cases of an infectious pyrexia is often a matter of much difficulty" and that "the more perfect our knowledge of all possible causes of such a condition, the greater will be our chance of arriving at a correct conclusion."

With the view of justifying the remarks I have made I will present in some of the observations which have been made on the combination of fever with cirrhosis of the liver, by some of the best authorities on hepatic disease.

McEwen states that at the onset there may be "a slight degree of fever," and further, "as the disease progresses acute hepatic extension manifests itself."

McEwen¹ also states that the disease "commences now and then in a more acute manner with febrile symptoms . . .

But in these cases there has probably been chronic mischief previously, and the acute symptoms have followed some imprudence in diet, or unusual excess in stimulants, or a chill."

In verification of this statement he quotes two cases in the appendix to his work. The first is that of a cab-driver who died after six months' residence in hospital, in which instance the "pulse varied from 80 to 100, and the temperature was on some days normal, but as a rule febrile, varying from 99.5° to

¹ 'Disease of Liver,' Sydenham Society's translation, vol. ii, p. 86.

² 'Disease of Liver,' p. 296.

101.5°. There were occasional perspirations, but these were never profuse and rigors were never present.

The other was that of a child, æt. 12, who had a pulse of 120 rising at the last to 140, and a temperature at first 102.3° falling towards the end to 95°.

It is perhaps noteworthy that both these cases were supposed by him to have arisen from a chill.

In another case, page 293, the temperature on admission was 101.2°, but subsequently "the temperature throughout was below normal standard."

In another case, page 302, a boy, æt. 9, the temperature on two occasions rose in the evening to 102.8° and 103.4° respectively, but it was usually under 100°. This was an undoubted alcoholic case.

At page 145, a case is recorded in which the temperature at night was usually 101°.

In all the other cases quoted by Murchison the temperature when recorded was either normal or subnormal.

Thierfelder¹ says: "In *exceptional*² cases, occurring chiefly in topers, there appear in the commencement of the disease severe periodical pains in the region of the liver, accompanied with great tumefaction of the organ, and *usually also with fever.*" Such attacks, which are wont to last some days, may be frequently repeated, sometimes at regular at other times at irregular intervals. We have here to deal, in all probability, with fluxionary hyperæmias, such as are also otherwise produced in this class of individuals by dietetic irregularities especially by excess in drinking."

I think it would be gathered from these quotations that the concurrence of fever with cirrhosis of the liver, of course apart from complications that might give rise to it in themselves, is to be looked upon as rare and "exceptional," and due to conditions that did not obtain in the instance I have recorded, since she was all the time in hospital and not subject to "dietetic irregularities;" further, the fever in her case can scarcely be described as "slight;" and, finally, it was, of course, not confined to the beginning of the case.

It appeared to me quite possible, from the ordinary teach-

¹ 'Ziemssen's Cyclopædia,' vol. ix.

² The italics are my own.

ing as to the relation of fever to cirrhosis of the liver, that it has been too often taken for granted that a febrile temperature was not to be expected in this affection, and that consequently extended observations had not been sufficiently commonly made.

In reference to this point I have collected from the clinical records of Guy's Hospital fifty cases of cirrhosis of the liver, uncomplicated by lesions which would of themselves cause fever, with the view of ascertaining the proportion of those attended by febrile temperature; and in the inquiry I have not failed to find confirmation of the remark I have just made as to the sufficiency of observations bearing upon this point.

Still, I have found such a number of carefully recorded cases (of which I propose to cite four examples) as are sufficient to show that the common statement is by no means correct.

Further, I would point out that the incompleteness in the records of temperature I have so often met with in reading the clinical reports, only serves to accentuate the conclusion I have arrived at, since I have been obliged to include many cases as "non-febrile" in which only one or two observations were made, and even a few in which none were recorded at all.

I will begin by quoting four cases in which a more or less careful record of temperature was made.

CASE 1 was under the care of Dr. Moxon in 1879.

The patient, a male æt. 33, was admitted on October 22nd and discharged on November 7th.

The record of the temperature runs as follows :

	Morning.	Evening.		Morning.	Evening.
Oct. 23.	99·8° ...		31.	98·8° ...	98·6°
24.	100·4° ...		Nov. 1.	98·9° ...	100°
25.	99·4° ...	99°	2.	99·6° ...	
27.	99·8° ...	98·8°	3.	98·6° ...	100°
28.	99·5° ...	98·7°	4.	98·6° ...	98·4°
29.	99·4° ...	100°	5.	98·6° ...	
30.	99·1° ...	99·5°			

CASE 2 was under Dr. Moxon in 1881.

The patient, a male æt. 32, was admitted on August 12th.

There is no record up to August 21st, after that date it runs as follows :

	Morning.	Evening.		Morning.	Evening.
Aug. 21.	99·4° ...		23.	103° ...	102·4°
22.	98·8° ...	98·6°			

Up to August 29th it varied from 99·8° to 99°, being usually lower in the evening.

	Morning.	Evening.		Morning.	Evening.
Aug. 30.	100°	...	17.	98·4°	...
Sept. 1.	98·6°	100·8°	19.	99·7°	...
2.	98·6°	...	20.	99·7°	...
3.	99·8°	100°	21.	99·5°	...
5.	99·4°	103·1°	23.	99·5°	...
6.	101·4°	102·1°	27.	98·8°	...
7.	100·4°	99·1°	28.	99°	...
8.	99·5°	99·4°	29.	99°	...
9.	99·6°	99·3°	30.	Paracentesis abdominis was now performed.	
10.	99°	99·2°	Oct. 1.	98·6°	...
11.	98·3°	98·6°	3.	98·8°	...
12.	97·6°	97°	4.	99°	...
13.	98·8°	...	5.	99°	103°
14.	98·4°	...	6.	101·4°	98°
15.	99°	...			
16.	99·2°	...			

Subsequently the temperature varied from 99·5° to 99°, and was occasionally normal. The patient died October 14th from hæmatemesis.

It is noteworthy that the temperature was on two occasions as high as 103°. The case has the advantage of having been brought to the test of an autopsy, which, however, revealed nothing that would account for fever apart from the cirrhosis.

CASE 3 was under the care of Dr. Pavy in 1883.

The patient, a female æt. 47, came into the hospital on February 26th.

There is no record of temperature up to March 18th, but subsequently to that date it was as follows :

	Morning.	Evening.		Morning.	Evening.
March 18.	101°	100·4°	28.	98·6°	100·8°
19.	100·4°	100·8°	29.	99°	100·4°
20.	100°	100·6°	30.	99·4°	100·8°
21.	100·6°	101°	31.	99·6°	99°
22.	100°	100°	April 1.	99·4°	99·8°
23.	99·4°	100°	2.	98·4°	98·4°
24.	...	100·4°	3.	...	99·6°
25.	...	100°	4.	98·2°	98·6°
26.	99·6°	100·8°	5.	98°	99·4°
27.	99·2°	99·4°	6.	98·8°	98·4°

The patient left the hospital on April 25th "much relieved."

350 *Enlarged, so-called "Hypertrophic," Cirrhotic Liver.*

CASE 4 was under the care of Dr. Pye-Smith during the present year.

The patient, a male æt. 53, was admitted on January 20th, and was discharged, "relieved," on March 20th.

	Morning.	Evening.		Morning.	Evening.
March 21.	99·4°			98·4°	98·4°
	99°	...		99·4°	97·8°
	99°	100°		98·6°	98·2°
	99°	98·6°		98·4°	99·2°
	98·4°	99°		98·2°	98·2°

For the rest the temperature was normal.

The result of an analysis of the records of the fifty cases may be stated as follows :

No mention of temperature	6
Non-febrile	<i>Few observations</i> 15
	<i>Many observations</i> 11
Febrile	<i>Few observations</i> 5
	<i>Many observations</i> 13

I have given as "non-febrile" several instances in which the temperature only rose on one or two occasions.

It would crowd this paper with unnecessary matter and greatly increase its tediousness if I were to give details of all the cases ; but the following statement may certainly be made with accuracy :

"That of the forty-four cases of cirrhosis of the liver in which observations of the temperature were made, eighteen were accompanied by an irregularly febrile temperature. It is right to add to this, that in eleven of these the observations were many and often continuous."

The result is imperfect for the reasons I have stated, but the deficiency only goes to prove my point more strongly, for the proportion is a high one, even if we reckon against it both the "non-febrile" and those in which "no mention" is made. But I think it cannot be doubted that many of the instances enumerated under the last two heads would have given a record of fever during some portion of their course, if continuous observations had been made.

A CASE
OF
EXTENSIVE CEREBRAL SOFTENING,
WITH DESCENDING SCLEROSIS IN THE LATERAL
COLUMN.

By G. N. PITT, M.A., M.D.

I AM indebted to Dr. Pavy for his permission to publish this case.

Emily S. M—, æt. 2, admitted August 14th, 1883, for right hemiplegia.

Family history.—Good.

Present illness.—Three weeks ago the patient had an attack of diarrhoea, ten days ago she had convulsions ; she has gradually been getting thinner and weaker.

On admission.—She lies quietly in bed, taking little notice of things going on around her. There is left facial paralysis, and the right arm is almost completely paralysed. There is occasional vomiting. Radial pulse weaker on right than on left side. The abdomen is retracted. The child has double optic neuritis, unequal pupils, and nystagmus. Pulse 108, resp. 24, temp. 98°.

August 21st.—During the past week the child has remained quiet, taking but little notice of surrounding objects, but this may possibly be due to blindness. It has vomited frequently. From time to time there has been rigidity of different limbs, and at times they have been paralysed. The paralysis of the

right arm was temporary. Occasionally a hydrocephalic cry has been noticed.

22d.—There is now left hemiplegia with left facial paralysis. The arms and legs have been strongly flexed, the thighs being drawn up to the abdomen and the fingers bent over the thumbs. Fits infrequent; frequent screaming; occasional vomiting.

September 12th.—There is now no facial paralysis; rigidity less marked: the optic neuritis is passing on to atrophy. The head seems to be increasing in size. The child seems better, and takes its food well. The odour from the child and from its muscles is extremely offensive, so that it has to be screened off from the other patients.

13th.—Left cornea rather insensitive; it is doubtful whether the child is blind.

October 1st.—There is now rigidity of both legs, but more especially the left, which is strongly flexed on the abdomen, the right not much less markedly so. The patellar reflexes are increased. The left arm is flexed and rigid, with incurved thumb: the right arm is a little rigid. There is left facial paralysis. The child lies on its left side paying no attention to anything. There is frequent vomiting, and the child is constantly crying. No anaesthesia. Subcutaneous abscesses have formed on the buttock and in the popliteal space; they have burst and are tending on the former to form bedsores.

November 1st.—No material alteration in symptoms. The optic discs are completely atrophied.

January 7th, 1884.—Slight facial paralysis.

14th.—Increased facial paralysis; discharge from left ear.

February 7th.—For two weeks there has been haziness of the left cornea, owing to the inability of the upper eyelid to move; it has been treated by warmth and protection, but has progressed almost to sloughing. The head has been for some time turned to the right. There is a discharge from the left ear. Limbs on left side rigid and strongly flexed, those on the right slightly so. The child has steadily emaciated until it has become extremely thin; it gradually got weaker, and died March 4th.

Post-mortem examination (by Dr. Mahomed).—The body was more emaciated than any I have ever seen; it was so wasted

that the superficial cervical plexus could be made out under the skin, and the junction of the rib with the costal cartilage was very distinct; the cartilage was much wasted.

The dura mater was not abnormally adherent to the brain or skull, but on the right side it is lined by a false membrane, blood-stained, reddish yellow in colour, and transparent, which peels off readily from the dura mater without adhesions, leaving a clean surface. Over the posterior part of this hemisphere and continuous with the false membrane is a blood-clot, black in colour, consisting of nearly a drachm of blood. This clot is recent, as it is not at all decolorised. There was a considerable amount of fluid in the arachnoid cavity; at the base the membranes look a little granular but contain no tubercle.

Brain 25 oz. The convolutions of the right hemisphere are wasted, and the surface is stained of a yellowish colour. A section through the centrum ovale majus shows the convolutions to be softened, and this softening extends inwards, but in a diminishing degree, so that only the outer parts of both the external and the internal capsules are affected. The extreme anterior part of the frontal, the posterior part of the occipital, the foot of the ascending frontal, the ascending parietal, and the angular convolutions have escaped the softening process. The ventricles, optic thalamus, corpus striatum, pons, cerebellum medulla, and spinal cord appeared healthy to the naked eye. The middle cerebral artery on the right side was thickened and almost occluded; it contained a thin thread of blood which seemed to mark its lumen.

Lungs.—Cheesy glands in the posterior mediastinum; tubercles under the pleura; the substance of the lungs contained grey tubercles. There was a cavity containing pus in each upper lobe.

Heart and blood-vessels normal.

Diaphragm, small intestine, mesenteric glands, spleen, and kidney contained tubercles.

In the liver were many tuberculous cavities containing pus.

The left tympanic cavity was full of pus and its walls were infiltrated. There was no direct implication of the membranes of the brain.

The choroid was free from tubercle.

Portions of the motor cortex, of the pons, and of the spinal cord in the cervical and dorsal regions were hardened in chromic acid and stained with carmine.

Cerebral cortex.—Structure indefinite; blood-vessels dilated, full of corpuscles, with granular material in the peri-vascular spaces. There were numerous leucocytes in the section.

Pons.—One side is stained in parts more deeply than in the corresponding part on the opposite side, due to an increase of neuroglia. This is most noticeable in the anterior part near the middle line.

Cervical cord.—There is a definite peripheral sclerosis of the neuroglia varying in depth from .25—.75 mm. Both the lateral pyramidal tracts are stained a deeper pink than the rest of the white matter, and there is an increase of neuroglia especially on the left side. The deeply-stained portion involves the crossed pyramidal tracts but does not extend to the periphery. On the left side very few axis cylinders remain, while on the right side there are plenty. There are numerous leucocytes in these areas, and scattered through the grey matter. The anterior columns are normal.

Lumbar cord.—There is a similar sclerotic peripheral band of neuroglia but no lateral sclerosis.

On admission the case was supposed to be one of tubercular meningitis, but as the child lingered on from week to week in an almost moribund condition with double optic neuritis, a diagnosis of cerebral tumour, probably tubercular, was made. While in the hospital the child emaciated greatly, so that after death the connective-tissue sheaths of the nerves were found free from fat.

At first the symptoms varied, and although paralysis of the right arm was at one time observed it soon disappeared, and the paralysis became definitely localised on the left side. These limbs became gradually rigid, the leg was at first extended and afterwards flexed. In this state the child lingered on for months, seldom moving, but often screaming out, helpless, and often to all appearances moribund.

The condition found post-mortem was most unusual. There was extensive softening of the middle part of the left cerebral lobe, and after a most careful search nothing was found to

account for it, although there was some narrowing of the lumen of the middle cerebral artery.

The presence of the optic neuritis on admission points to the occurrence of the softening previous to this time ; it was soon followed by rigidity.

There was well-marked descending sclerosis of the crossed pyramidal tract in the cervical region of the cord on the left side and a much earlier condition of the same on the right side. There was sclerosis in the motor tract on the right side of the pons. The lumbar cord showed no descending sclerosis. The peripheral sclerosis was probably due to some meningitis.

Most of the organs of the body were tuberculous and there were cheesy glands in the mediastinum. It is just possible that there had been some tubercular thickening of the middle cerebral artery leading to anæmia and softening of the brain, but against this view there are several points.

There was no evidence of tubercular meningitis, and it is unlikely that a large vessel alone would be affected while the smaller ones, so far as they were examined, appeared to have escaped. Moreover, had the softening been due to occlusion of the main trunk of the middle cerebral artery, the optic thalamus and corpus striatum would have been softened, whereas they escaped, as did also the lower part of the ascending frontal and parietal convolutions. There was no evidence of the softening being due to syphilis, to thrombosis of the sinuses, or to heart disease.

Dr. Gowers states that thrombosis of healthy cerebral vessels in young children occasionally occurs, but in this case none was discovered after careful examination.

We are led, therefore, to the conclusion that we may get in a child two years old extensive cerebral softening without any discoverable cause, which may still permit life to continue for six months.

Dr. Taylor recorded a case in the ' Guy's Hospital Reports ' for 1879 of an extensive gelatinous degeneration of the brain with descending sclerosis of the cord in a boy aged six. No cause was discovered, but the case differs from this one in that it was not a softening of the brain substance but a degeneration into a brown jelly-like material.

In the last volume (vol. xxvi, 3rd ser., 1882) Dr. Hale White

recorded a case of symmetrical softening of the corpora striata with descending sclerosis in a boy aged 6, for which also no cause could be found.

OBSERVATIONS
ON THE
DETERMINATION OF THE HEARING POWER.

By W. LAIDLAW PURVES, M.D.

DESPITE the numerous proposals and methods of testing the acuteness of hearing in affections of the ear, the instruments to which surgeons almost exclusively confine themselves for that purpose are the watch and the tuning-fork. That with such, a complete examination cannot be made will be admitted by all who have paid attention to the subject. The want of an instrument by which the intensity of speech could be regulated, and of a method of determining the accommodation of the ear, is still felt. That something in this direction may be arrived at by a modification of the telephone we may reasonably hope. Meantime I wish to direct attention to one or two points regarding the determination with the present instruments at our command.

In testing with the watch or other acoumeter of known intensity, the acuteness of hearing is determined by measuring the greatest distance at which the vibrations emitted by the instrument are heard through the air. Should that correspond to the distance already determined to be that of normal hearing, the hearing through the usual sound-conducting apparatus is considered normal. Should it be greater or less it is + or — and the amount is noted. The watch may also be employed

in roughly testing the power of audition through the cranial bones by mapping out the positions on the cranium at which a certain sound is heard, but the tuning-fork is a much more convenient instrument for this purpose, as by it the intensity of the sound may be increased at will, within limits which suffice for our purposes, and the instant at which the perception of vibrations ceases can be more exactly stated.

The duration is here determined by striking a prong of the tuning-fork against some hard substance, and placing the extremity of the handle on some part of the cranium, the teeth, vertex, or mastoid process, usually noting the weakest vibration perceived by the patient, and then measuring the time the note continues to be heard by a normal ear after the transference of the fork to a corresponding part of the observer's head. This does not give us an exact result, but for usual clinical purposes it is sufficiently so. If the fork be transferred on the cessation of the auditory perception from the bone, and placed close to the external opening, the sound will continue to be heard for some time longer should the conducting organs be in a normal condition. It is to the length of time which a normal ear continues to hear these aurally transmitted vibrations that I wish to draw attention.

The duration of the mental after the mastoideal hearing is influenced by several factors, viz. the position of the fork on the cranium, the note of the fork, the size of the fork, and probably by other agents to which we need not here refer.

From the teeth the waves are conveyed to the auditory nerve, probably better than from any other part, but as they are not always present nor in a condition to be placed at the surgeon's service, and as the end to be attained can be reached more conveniently from the mastoid, it has been chosen as the spot through which perosseal audition is conveyed.

Causing a C = 512, with prongs $4\frac{1}{2}$ in. in length, to vibrate, and placing it on the portion of the mastoid at which it is best heard, and then on the cessation of the perception through the osseous structures transferring it to the mental orifice, I find that it continues to be heard by me for thirty seconds. On determining this with what are considered normal ears, I find this ranges from twenty-five to thirty-five seconds, depending partly on the age of the patient. Can we use this ratio of

meatal to mastoideal hearing in any way for the more exact localisation of affections, and the apportioning of the amount of loss to its proper seat, where both the conducting apparatus and the nerve are implicated?

If the tuning-fork ceases to be heard by the patient and a healthy observer at the same moment, we conclude roughly that the acoustics of both are equally sensitive to that tone. Should, however, the patient hear vibrations to which the observer's acoustic does not respond, we conclude that either the acoustic of the patient is more sensitive or that there is some additional reflection by some hindrance to the dispersion of wave-sounds from the ear. The number of seconds during which the vibrations are heard after they are lost to a healthy ear is noted as + per mastoid. To determine to which locality we must assign this, the duration of the meatal hearing after the cessation of the mastoideal is determined. Should this also be above the normal standard a hypersensitiveness of the acoustic is determined. But should the meatal hearing be of less than the normal duration after the mastoideal has ceased, we conclude that vibrations to which the acoustic ought to respond through the meatus are not of sufficient strength to set in motion the conducting apparatus, and we have thereby a gauge as to the amount of obstruction to the passage of vibrations through the normal channels.

Should the acoustic be deficient but the meatal hearing be in the same proportion to the mastoideal, the conducting media may be taken as normal, and the deficiency of the acoustic taken by the number of seconds during which a healthy nerve detects the pulsations after the removal of the fork to the mastoid of the observer. Or, by closing the meatus of the patient, and so causing an increase to and perception of the vibrations by the deficient acoustic as long as they are detected by a healthy nerve through the mastoid, the difference of the meatal hearings thereafter will give the deficiency.

To make these and many such like determinations conveniently, a stop-watch, a large fork, and an apparatus formed to cause the fork to be heard by the patient and by the observer at the same time and with equal pressure, are required. By these the physician may determine the functional power of the acoustic, and without visually examining the meatus and cavity assign in a general way the loss, if any, to its proper position

The determination, as generally carried out, must lead to false conclusions if the difference of the meatal from the mastoidal hearing, and the ratio of the two are not kept constantly in view. We require numerous observations before we can fix a standard of the two hearing powers at different ages and of the proportion of one to the other, but having gained these, we shall have added another step to the exactitude of our diagnosis. That many acoustic changes which are not detected occur in early stages of different nervous diseases I feel certain, and I would assure neurologists that one hour's application would enable them to add a valuable indication to their means of detection of early abnormal deviations.

A CASE OF HYDROPHOBIA, IN WHICH THE CONDITION OF THE LARYNX WAS OBSERVED DURING A SPASM.

By G. N. PITT, M.A., M.D.

I AM indebted to Dr. Wilks for his kindness in permitting me to publish this case, which is reported by Mr. F. B. W. Phillips.

J. R—, æt. 46, was admitted October 29th, 1888, with difficulty in swallowing. On May 15th the patient was bitten by a favourite dog. The animal is said to have had a fight with another dog, to have returned home in an excited manner and attacked its master, biting him first on the right side of the face at the angle of the jaw, and then biting his trousers without wounding his leg. At the same time the animal bit the patient's son, a man æt. 26, but it is not known which was attacked first.

Hardly any notice appears to have been taken of the incident at the time, the dog was beaten and was never seen again. The history was only obtained by cross-examining the wife. The man had no idea that he had hydrophobia. The patient was fond of animals and kept several dogs and cats.

He had been subject to bronchitis and slight attacks of rheumatism for several years, but his wife says that since he was bitten he had been "ailing."

Nothing, however, was particularly noticed until October 27th. In the evening he complained of earache on the right side and of pain from the front to the back of his neck. He

had supper and slept fairly well, though in addition to the earache he now developed a sorethroat.

On the 28th he felt a little better and ate a good dinner. About 10 p.m. a feeling of choking came on with a "spasm."

According to his wife's account, he threw his head back, opened his mouth, called for more air, and felt choked. He was unable to swallow though he tried to do so, and a spasm was excited when drink was brought within six inches of his mouth. The spasms also occurred, she said, independently of the sight of fluid and at intervals of about a quarter of an hour.

He was quite unable to sleep and kept walking about all the night; he perspired freely and was feverish. There was no pain except during the spasms. This condition continued, and this morning he was seen by a medical man who ordered him some medicine, which the patient was unable to swallow. No food or drink has passed his lips since dinner yesterday. The bowels were opened on the 27th.

Patient was admitted into the ward as no private room was vacant.

He is a powerful, tall, well-built man, weighing about fifteen stone, with dark hair and clean shaved upper lip and chin. His countenance expresses no anxiety, he is quiet and rational, and feels comfortable except for the dryness of his throat. He expresses no fear with reference to his inability to swallow. When asked to try, he put some milk to his lips and after a series of convulsive efforts managed to swallow a drachm or so; he said the sensation was that of having his gullet drawn out of him. On repeating the attempt, directly the mug was within a few inches of his mouth, his facial muscles contracted and the mug was thrust against his clenched teeth.

He permitted the house physician, Mr. Pitt, to examine him with a laryngoscope. The mirror was cautiously introduced and a good view of the larynx obtained. On the second occasion a spasm occurred and the vocal cords, instead of meeting in the middle line as was expected, separated widely and remained so for some seconds. The larynx looked healthy. The tongue and palate were furred. Temp. 99°, pulse 93, resp. 15.

He passed a comfortable night and slept several times. He had no pharyngeal spasms but complained of "cramp in his

stomach" occurring frequently. The nurse says he sat up in bed and took long, deep inspirations. He was very anxious his wife should stay with him. When the nurse brought a lamp to his bedside no spasm was excited. He passed his water without difficulty; his bowels were opened once.

He was allowed slices of lemon to suck and had nutrient enemata which contained also chloral hydrate gr. xx and bromide of potassium gr. xx at 11.30 p.m., 3 a.m., 6 a.m., and 9 a.m. He had nothing to drink.

October 30th.—His employer came to see him and said he looked the same as usual. He also said that the patient had had no spasms during the three quarters of an hour he was with him on the preceding morning.

Noon.—Patient spat out a pellet of thick mucoid material, said his mouth was very sticky, and was constantly rubbing it with his handkerchief.

1.30 p.m.—Pulse 80, full and strong; resp. 18, temp. 99.2°. Pupils rather dilated, skin warm and moist; no spasms. He complained at first of the noise in the ward but is now used to it. Nutrient enema.

6 p.m.—About 4 p.m. he walked across to a private room. Bowels opened once. At first he expressed himself pleased with the room, but afterwards his manner changed and he was very anxious to get away. He is now very restless, complains that he is thirsty, and that his mouth is sticky; expectoration very viscid, tongue not dry. On giving him a piece of ice to suck, he took a deep inspiration and got it in his mouth, working his facial muscles violently, but he appreciated the coolness in his mouth. Pulse 126, temp. 100.2°. He had some glycerine and lime-juice to moisten his lips, for which purpose he sat up and dipped in his finger in an abrupt and agitated manner, but rubbing his lips did not induce a spasm. Nutrient enema.

7.30 p.m.—Patient is extremely nervous, complains that he has great oppression across his chest and that he is being starved, entreats one to poison him at once instead of suffocating him slowly. He was with difficulty pacified. Shortly afterwards, however, he got out of the room in spite of the nurse, and nothing would induce him to return to the room, where he said he felt he was being suffocated. He had hallucinations of sight, fancying

he saw creatures trying to murder him. He was with difficulty got back to bed and became so extremely and maniacally violent that it was necessary to strap him down. Expression one of extreme terror; pupils widely dilated. He expectorated a viscid secretion in all directions and had such severe pharyngeal spasms that it was necessary to give him chloroform for ten minutes at 8.40. Resp. 44, pulse 140.

9 p.m.—Breathing noisy, much exhausted after his violent struggles. A few drops of chloroform given occasionally to keep him quiet.

9.30.—Injectio morph. (gr. $\frac{1}{4}$) et atropiæ.

11.—Patient is now quiet again; spasms of diaphragm and pharynx occasionally when speaking. Chloroform discontinued.

12.15—Chloroform given and nasal tube passed; this at first went easily into the trachea and excited no spasm of adductors of vocal cords. The tube was then passed into the stomach and—

Milk Oj,

Egg j,

Brand's Essence ʒss ,

Potassium Bromide gr. xxx,

Chloral Hydrate gr. xx,

were given. Enemata were stopped.

Pupils smaller, temp. 102.4° , pulse 129; inj. morphiæ (gr. $\frac{1}{4}$) et atropiæ.

October 31st, 1.30.—Cheynes Stokes' breathing, some respirations being deep, noisy, and spasmodic.

7 a.m.—Has had a quiet night and slept part of the time. Occasionally incoherent in his talk.

9 a.m.—Has been talking about himself and various things quite rationally. Violent spasms suddenly recurred. Chloroform administered occasionally as required. Temp. 103° .

10 a.m.—Fed under chloroform with nasal tube. Inj. morphiæ (gr. $\frac{1}{4}$) et atropiæ; pulse 136, temp. 105.6° . Patient is very restless and has some dyspnœa. No priapism at any time.

Noon.—Pulse failing, general râles all over chest. Unconscious. Pupils dilated. Eight ounces of urine drawn off containing some blood; it was smoky in colour. That passed the preceding night was normal.

1.30.—Circulation became feebler and patient died. Temp. 103.6° .

2.30.—Temp. 104.2°.

Autopsy.—Spinal cord: small hæmorrhages in the hinder part of the left anterior cornu in cervical region, another in the right anterior cornu in dorsal region. Bony plates on posterior surface of cord. Numerous puncta cruenta scattered through cord in both grey and white matter. Vessels of pons and brain congested. Lungs œdematous. Other organs normal. Nothing further was discovered by microscopical examination.

There are several points worthy of note in this case. Granting that the disease is, with our limited present experience, necessarily fatal, the treatment was successful in that the spasms were kept in abeyance for twenty-four hours by avoiding any food or drink by the mouth and feeding the patient by nutrient enemata with a scruple each of potassium bromide and of chloral hydrate every three hours. There was then an outbreak of maniacal violence which was subdued only by inhaling chloroform; and by administering it occasionally the patient was kept quiet and free from the distressing spasmodic attacks until the time of his death, except once when the spasms were severe and these lasted only a few minutes until chloroform was administered.

A few hours before his death the patient was quite rational, and was talking without any distress. After the pharyngeal spasms were once severe the patient secreted a very viscid saliva which annoyed him; he was constantly trying to spit it out, but it stuck to his mouth. I was fortunate enough to get a good view of the larynx during a spasm, and to be able to observe that the spasm affected the abductors and not the adductors as has always been supposed. That this was the case was also corroborated by the ease with which the nasal tube passed down the trachea without being grasped by the vocal cords and without exciting a spasm of the adductors as would usually be the case.

During the last ten years there have been five cases of hydrophobia at Guy's Hospital, and all have ended fatally. In 1877 there were three cases, a girl of five, a boy of fourteen, and a man of thirty-four. In 1878 there was a man of thirty-six, whose mania took quite a religious type. In all there were the pharyngeal spasms. Dilated pupils were noticed, and the

men were constantly spitting as this man did. They were treated with curare, but the violent spasms were not kept in check.

We have to recollect that our treatment is not only to benefit the patient when possible, but also the friends, and in but too many fatal diseases this latter is all we can do. It is really a great thing to be able to keep the maniacal violence and distressing spasms of the patient under control. Our sheet-anchor for this purpose is chloroform, a few drops of which may be administered occasionally as required, and more may be given when feeding the patient by the nasal tube. Bromide of potassium and chloral in doses of two to three drachms daily may be given with nutrient enemata, and occasional injections of morphia, as required, to assist the sedative effect of the chloroform.

Whether a new era in treatment has been opened up by Pasteur's experiments it is yet early to say, but as so many people are bitten by mad dogs without developing hydrophobia it will be impossible in any individual case to prove that the treatment has averted the disease unless the symptoms have already developed, in which case we can hardly expect even an inoculation with minimised virus to protect the patient from further mischief.

**ON A CASE OF GASTROSTOMY,
IN WHICH THE ARTIFICIAL OPENING WAS
SUBSEQUENTLY CLOSED.**

By N. DAVIES-COLLEY, M.C.

A GREAT impulse has of late years been given to the treatment of impermeable stricture of the œsophagus, firstly by Verneuil's introduction of a method of gastrostomy by which the surface of the stomach is closely adjusted to the abdominal parietes, and, secondly, by Mr. Howse's plan of postponing the opening of the stomach itself until proper adhesions have been established. Many cases of traumatic, cancerous, and other forms of stricture of the œsophagus have thus been successfully treated in Guy's Hospital and elsewhere, but as far as I can learn, none have as yet been fully recorded in which the operation has been followed by such amelioration in the condition of the stricture as to allow of the subsequent closure of the artificial opening. Judging from the improvement in strictures of the urethra and rectum which often results from analogous operations, such as the form of perineal section in the membranous urethra, introduced by Mr. Cock, and colotomy in the case of rectal disease, we should anticipate that a similar improvement would sometimes be met with in cases of œsophageal contraction.

By the performance of gastrostomy the ulcerated and inflamed tissues are placed completely at rest, for they are no longer

subject to the continual irritation produced by the passage of food, and by the decomposition of those particles which remain in the pouch that tends to form above the stricture. Moreover, this dilated condition is in itself a serious obstacle to the employment of instruments, and it is reasonable to expect that after the accumulation of food at this point has been prevented by feeding the patient through the opening in the abdominal wall, the parts above the stricture will sink to their ordinary dimensions, and allow the use of bougies to be resumed with success. In the case which I shall now narrate, it will be seen that after the successful performance of gastrostomy for the relief of cicatricial stricture following ulceration, it was not even necessary to have recourse to instrumental dilatation, but that a rest of eight months had caused such a diminution of the disease that I was able to close the artificial opening, and that this was followed by a steady and permanent improvement of the patient's condition.

M. A. W—, æt. 34, single, cook, was admitted into Charity Ward under Mr. Cooper Forster on January 22nd, 1879, and subsequently transferred to my care.

She has heard of no tumour nor cancer in any member of her family. Her mother died of consumption. As long as she can remember she has suffered from sorethroat, occasionally accompanied by loss of voice. Eight or nine years ago she had cough and hæmoptysis, and one year later abscesses on the right side of the neck. She first had difficulty in swallowing five years ago, and this difficulty has been gradually increasing ever since. Two years ago she spent three weeks in the City Road Hospital for Diseases of the Chest, and the next year she was in St. Bartholomew's Hospital for six weeks, where unsuccessful attempts were made to pass bougies. Latterly she has been unable to swallow anything but fluids, and these only very slowly. At the same time she has become much weaker, and she is now unable to do her work. On the morning of her admission she was seen by me in the out-patient department. I passed a bougie about one third of an inch in diameter with some difficulty a foot beyond the lips. There was a considerable obstruction at the level of the upper end of the sternum. As soon as I withdrew the instrument, she complained of great pain, and as she also appeared much collapsed she was taken in.

On admission she was fairly nourished, though she stated that she had been much stouter. Her tongue was scarred and glazed, and there was a band of very white and prominent fur in the centre. She was complaining of severe pain in the chest. A subcutaneous injection of morphia was given, and she was kept in bed and fed by nutrient enemata. In a few days she had quite recovered from the pain and shock caused by the passage of the bougie, and could swallow better than she had done for some time before. A fortnight later she could take fish and bread, but no meat. Subsequently the difficulty of swallowing again returned, and she was obliged to restrict herself to fluids.

As the condition of the tongue made it probable that she had had syphilis, a mixture containing 10 grains of Iodide of Potassium and 80 minims of Liquor Hydrargyri Perchloridi was ordered to be taken three times a day; but it had to be discontinued, for within two days an eruption of large bullæ broke out over the upper part of her body, her face, and arms. She stated that the same rash had formerly followed the administration of a similar medicine.

On March 13th an unsuccessful attempt was made to pass a bougie. She afterwards coughed up small quantities of blood, and her strength appeared to be failing.

Mr. Cooper Forster having transferred the patient to my charge, I proceeded on March 22nd to operate with the object of forming an artificial opening into the stomach.

Chloroform having been administered, I made an incision three inches long, parallel to the left costal margin, at a distance of half an inch below it, and with its upper end between two and three inches from the linea alba. The fat was nearly half an inch thick. The muscles also were well developed, especially the transversalis. On opening the peritoneum, the stomach was at once found, and the attachment of the great omentum was visible at the lower end of the wound. After drawing down the stomach, I united it to the abdominal wall over a circular space nearly three inches in diameter, by silk sutures passed through the parietes on the one side, and the serous and muscular coats of the stomach on the other. I then drew the margins of the skin to the surface of the stomach by silver sutures which formed an inner circle one inch in

diameter. The hæmorrhage was very slight. No vessels had to be tied or twisted. The spray was used, and oiled carbolic gauze was subsequently applied.

No peritonitis followed. She was fed chiefly by enemata of milk and beef tea, but was able to take small quantities of milk by the mouth. She complained of feeling sick, but did not vomit.

On the 27th, five days after the operation, I made an incision three-quarters of an inch long in the wall of the stomach, and inserted a vulcanised india-rubber tube about half an inch in diameter. Her condition was at that time but little worse than it had been immediately before the operation. During the twenty-four hours before I opened the stomach she had been able to swallow a quarter of a pint of beef tea and half a pint of milk. She had, however, become unable to retain the nutrient enemata. Four hours after the stomach was opened I began to feed her through the tube with warm milk, and from this time all her nourishment was given her in this way. Her power of swallowing for a time ceased. The chief inconvenience she suffered was from the escape of food by the side of the tube.

On April 18th I took away the last of the sutures, and allowed her to sit up in bed.

May 7th.—She was able to walk the length of the ward.

31st.—She has gained flesh since the operation and is much stronger than she was three months ago. She takes some liquid food by the mouth, but from time to time her œsophagus becomes so occluded that she cannot even swallow her saliva.

At the end of July she was much thrown back by an attack of erysipelas round the margins of the opening, and she was for some time unable to leave her bed, partly on account of weakness, but chiefly because the food ran out when she assumed the upright posture. She also began to take her food by the mouth again. It consisted chiefly of liquids and bread and butter.

October 10th.—There is a good deal of excoriation round the wound, and great pain is felt there when the fluid escapes. This is chiefly when she is up and walking about, so for some time she has been obliged to keep to her bed. To-day the tube was altogether removed, and but little fluid has escaped.

18th.—The inner surface of her lower lip is covered with mucous patches.

27th.—Still complaining of the escape of fluid.

November 4th.—Chloroform administered. The opening in the abdominal wall is now about one third by one quarter of an inch in dimensions. As the surface of the adjacent skin was excoriated and partly covered with granulations, I did not make any incision, but inserted two pairs of button sutures half an inch from the edges of the opening, and so brought the raw surfaces together.

13th.—Fluid began to escape again through the opening.

21st.—Both the sutures have been removed; the opening is now much narrower. Fluid does not escape so often as before they were put in, but the discharge is quite as copious when it does occur.

December 2nd.—Chloroform was again administered, and the edges were pared in a bevelled direction at the expense of the skin, so as to leave all round a raw surface a quarter to one third of an inch broad. Three pairs of button sutures were used, and also two wire sutures for the more accurate approximation of the margins. The parts were thus considerably puckered up, and a deep furrow was formed at the lower extremity of the wound.

9th.—Good apposition.

23rd.—The second pair of buttons was removed.

26th.—The third and last of the pairs of buttons was removed. A little flatus has escaped since the last notice. She feels comfortable, and was well enough yesterday, Christmas Day, to take a little plum pudding. She can sit up in the bed.

On January 13th, 1880, she went out with the wound quite healed.

On July 24th, 1883, I saw her again. She told me that after she went out there had been a little discharge from the wound until a bit of wire came away. About Christmas, 1881, her power of swallowing still further improved, and she began to take meat and solids well, provided that she carefully masticated them. There has been no stomach pain or dyspepsia. Eighteen months ago she married, but she has had no child since her marriage. The scar left by the gastrostomy wound

is not at all puckered nor drawn in. It is quite smooth and would easily escape notice.

Her tongue has along the centre a fairly natural appearance, but the sides are glazed. The immediate occasion of her coming to see me was a severe attack of eczema and scabies, from which she soon recovered.

Remarks.—The first point which occurs to one in the consideration of this case is to determine what were the cause and nature of the original disease. The patient could give us no history of the swallowing of any corrosive material, and her age and appearance contra-indicated the presence of malignant tumour. The only clue we could find to explain her condition was in the evidence of extensive ulceration presented by her tongue. Subsequently we ascertained that she had had an illegitimate child, so that it was not improbable that she had been the subject of syphilitic infection. This probability was further strengthened during her stay in the hospital by the occasional eruption of mucous patches on the inside of her lips. She was peculiarly sensitive to the action of iodide of potassium, as it brought out large crops of vesicles and bullæ within a few days, and from the fact that she had previously had similar rashes after medicine it was evident that she had already been treated with the same drug, and probably on account of a similar diagnosis.

Very likely the patient might have been spared much of the inconvenience and pain which arose from the escape of food and gastric juice through the abdominal opening, if a smaller incision had been originally adopted. I am disposed also to think that Mr. Howse's plan of cutting through the parietes nearer to the middle line, and dividing the rectus longitudinally, is a good one. It is probable that the muscular fibres of that muscle when thus separated may form a sort of vertical sphincter, which may tend to close the orifice and prevent the escape of fluids during coughing and other violent exertions of the abdominal muscles. In one successful case now under my care I have adopted these two precautions, and although three months have elapsed since the stomach was opened, and the patient is now able to walk about, there has as yet been no trouble from the escape of the contents of the stomach.

I cannot say how much the contraction of the œsophagus has been diminished. The former attempts to pass bougies

caused such great pain, and the symptoms of collapse were so urgent, that I have not ventured to repeat the attempt. Judging, however, from the freedom with which she has been able to swallow meat and other solid food, I should expect to find that there is now a free passage into the stomach.

Mr. Bryant has kindly permitted me to add to this paper the further progress of a case which he published three years ago in the '*Lancet*.'¹ Although no operation was performed to close the gastric fistula, the improvement in swallowing obtained by the temporary rest would appear to have been nearly as great as in my case. The gastrostomy was performed in August, 1880, upon a girl, æt. 22, on account of a stricture caused by the attempt to commit suicide by swallowing half a wineglassful of sulphuric acid in the previous January. She could only swallow fluids, and with such difficulty that it took her an hour to get down half a pint of milk. Five days later a small opening, not more than one eighth of an inch long, was made into the stomach, and from that time she was fed through it. In three months she had gained 19 lb. in weight. The rest of the history I will repeat in Mr. Bryant's own words: "The girl married and was confined after a natural labour on August 14th, 1883. I saw her on May 6th, 1884, when she was in good health, and the gastric fistula was open. She could then drink freely, and took all soft food well; even meat when very finely divided. She said improvement in swallowing began about eighteen months after the operation and had steadily continued."

¹ '*Lancet*,' vol. i, 1881, p. 572.

ON COLLES'S FRACTURE.

BY R. CLEMENT LUCAS, B.S.

- I. EXPLANATION OF THE PAIN ON THE INNER SIDE OF THE WRIST AND HAND.
 - II. HOW THE POSITION OF THE ANNULAR LIGAMENTS DETERMINES THE EFFECT OF MUSCULAR ACTION IN MAINTAINING OR REPRODUCING THE DEFORMITY.
 - III. FRACTURE OF THE STYLOID PROCESS OF THE ULNA, AND RUPTURE OF THE TRIANGULAR FIBRO-CARTILAGE ARE FREQUENT COMPLICATIONS.
 - IV. EXPLANATION OF THE FREEDOM OF MOVEMENT REGAINED AFTER A TIME, THOUGH THE DEFORMITY REMAINS PERMANENT.
 - V. EXPLANATION OF THE ABSENCE OF CREPITUS.
 - VI. OWING TO SHORTENING OF THE RADIUS THE HEAD OF THE ULNA MAY BE FOUND BELOW THE LEVEL OF THE ARTICULAR SURFACE OF THE RADIUS AND THE ARTICULAR SURFACE ON THE ULNA FOR THE SIGMOID CAVITY OF THE RADIUS MAY BE CORRESPONDINGLY RAISED.
 - VII. A CASE IN WHICH THE LOWER FRAGMENT WAS ANTERIOR TO THE UPPER.
 - VIII. A DESCRIPTION OF COLLES'S FRACTURE.
-

THOSE who read Colles's original description of fracture of the lower extremity of the radius,¹ written in 1814, cannot fail to be struck with the clearness and conciseness of the account there given of the clinical signs and symptoms of the injury. It is a description in three pages, to which the mass of subsequent literature has added nothing of importance, because

¹ "On Fractures of the Carpal Extremity of the Radius," by A. Colles, M.D., Edin. Med. and Surg. Journal, 1814.

nothing of the kind was there omitted. But the writer had not at the time had an opportunity of dissecting a specimen, and all subsequent discussion has turned upon the cause of the deformity, the position of the fragments, or the appropriate treatment.

In March 1877, I had an opportunity of dissecting a case of Colles's fracture of long standing, wherein the deformity had been permanent and had remained a permanent disfigurement. The drawing of the hand accompanying this paper was made at that time. Since then I have had the good fortune to examine and dissect two more specimens. It was my original intention to write an extensive article on the subject, and with that object I read much that had been written by others. The paper was first written three years ago, when an illness prevented its completion. A similar fate again awaited it and prevented its appearing in the last number of the 'Reports.' I have now made aside all I compiled from the works of others, but the article has become bulky and discursive, and I desire to keep as closely as I can to the points it is in my power to establish.

1. Explanation of the pain on the inner side of the wrist and hand.

The pain on the inner side of the wrist has been noted by various writers, and has generally been explained by damage done to or stretching of, the internal lateral ligament.¹ It is increased by pressure below the ulna and can often be traced to the interval between the ring and little finger or into those fingers. The plate accompanying this paper, made from an actual dissection, clearly shows that this pain is caused by the projecting head of the ulna displacing and stretching the dorsal branch of the ulnar nerve, which lies immediately in contact with it. In my dissection a thick-walled bursa (rather exaggerated in the lithograph) was found developed over the head of

¹ "When pressure is made immediately below the head of the ulna considerable pain is produced, for the internal lateral ligament is put upon the stretch by the displacement of the carpus and lower fragment of the radius." (Prof. R. Smith, *Fractures and Dislocations*.)

the ulna. The fracture had happened nine years before the patient's death, but the angular displacement of the nerve was well shown. Stretched like a bowstring by the projecting ulna, the dissection explains how pressure immediately below the head of that bone would intensify the pain.

II. How the position of the annular ligaments determines the effect of muscular action in maintaining or reproducing the deformity.

Since Voillemier¹ suggested that the deformity and absence of crepitus were to be explained by the impaction of the upper fragment into the lower, a battle has raged between his followers and those who maintain that muscular action is the sole cause of the deformity. So far as the evidence of impaction has been drawn from cases examined after union had taken place, we must regard it as inconclusive; for, as Professor R. W. Smith² pointed out, a section through the bone in such cases will almost always yield an appearance of impaction, owing to the new bone developed for repair encasing the upper fragment. On the other hand, unless we are to doubt the personal observations of such good observers as the late Mr. Callender³ and Mr. Erichsen,⁴ impaction has been found in recent cases, and to such an extent as to be with some difficulty detached after death.

Admitting that impaction does take place in a certain number of cases, it must not be lost sight of that muscular action is alone sufficient to maintain and reproduce the deformity. It will be well to quote a passage from Colles's memoir, which I do not find alluded to in any of our text-books, but which it is certain that keen observer did not write without having satisfied himself of its accuracy: "If the surgeon lock his hand in that of the patient, and make extension, even with moderate force, he restores the limb to its natural form, but the distortion of the limb instantly returns on the extension

¹ 'Archives générales de Médecine,' March, 1842.

² 'Fractures and Dislocations,' 1847, p. 160.

³ 'St. Bartholomew's Hospital Reports,' vol. i, p. 283.

⁴ 'Science and Art of Surgery,' 6th ed., vol. i, p. 326.

"being removed." What muscular action will account for the reposition of the distal end? No one will suggest that the fragment became a second time impacted. No other force than muscular action is available.

It is my belief that those who have referred the deformity to the action of muscles have not clearly explained the mechanism by which it is brought about. Colles himself refers the dorsal displacement of the lower fragment to the action of the tendons of the extensor muscles of the thumb only. Smith adds the supinator longus, and argues for the direction of the articular surface downwards and backwards and the raising of the styloid process so that the articular surface is directed somewhat outwards, as follows:—"This twofold displacement is the result of the action of the supinator longus upon the one part, and of the long extensors of the thumb upon the other; the latter, as soon as the resistance of the carpus is removed by the occurrence of the fracture, draw the lower fragment of the radius, with the carpus, towards the side of extension, thus directing the articulating surface of the radius upwards¹ and backwards, while the former muscle, viz. the supinator longus, besides supinating the lower fragment, also elevates the styloid process, giving the carpal surface an inclination outwards."

The loss of balance between the muscles on the anterior and posterior surfaces of the wrist which follows fracture of the lower end of the radius, and enables the latter muscles to draw back the separated extremity, depends on the relative positions of the anterior and posterior annular ligaments. The posterior annular ligament crossing the ends of the radius and ulna binds down all the extensor tendons, save two, to the dorsal surface of the radius. Consequently, when the radius is fractured immediately above the ligament, not only the supinator longus and extensors of the thumb, but in addition the extensor carpi radialis longior, extensor carpi radialis brevior, extensor communis digitorum, and extensor indicis will all tend to drag back the fragment, and give a backward direction to the articular surface. On the other side, owing to the anterior annular ligament being on a lower level, running across the bones of the carpus and being unattached to the radius, the flexor tendons have no direct influence upon the

¹ "Upwards" in Smith's book (op. cit., p. 140) is a misprint for "downwards."

fragment and the extensor tendons are consequently left unopposed. This arrangement accounts for the recurrence of the deformity, when extension upon the hand, sufficient to overcome it, is withdrawn.

Muscular action is alone adequate to produce the deformity. As a rule, however, the direction of the force, transmitted through the lower end of the radius from a fall on the palm, first determines the displacement; and muscular action, which coincides in its result with the effect of the external violence, maintains or reproduces it.

I am fortunate in being able to publish, through the courtesy of Mr. Couper, an important case showing that post-mortem rigidity may bring about the characteristic deformity, which was absent during life, owing to paralysis of the limb.

The case is of especial value, since it was seen during life by that excellent clinical observer, Mr. J. Hutchinson, and dissected after death by so careful and exact a surgeon as Mr. Couper.

The date at which the dissection was made, April 5th, 1860, in no way detracts from its importance, since the following notes were all written out at that time. It is only to be regretted that they should have been so long withheld.

The subject of the injury was an old woman, believed to be over seventy years of age, who was admitted into the hospital three days before in a state of insensibility from injuries just received. The right arm and leg were found paralysed as a consequence of the brain injury. A fracture of the right wrist was examined by Mr. Hutchinson during the paralysed state of the limb. He states there was some swelling at the wrist, but none of the usual deformity so characteristic of Colles's fracture. He could crepitate with a small fragment behind at a spot in a line continuous with the middle line of the interosseous space. At first he doubted the existence of fracture, and then thought the radius split longitudinally.

The dissection was made a few hours after death, when the post-mortem rigidity was complete. Besides the fracture at the wrist the patient was found to have a fracture of the base of the skull.

There was a rounded prominence of muscles and tendons immediately above the anterior annular ligament extending

upwards about two inches and gradually subsiding into the normal contour of the forearm. There was a hollow on the posterior aspect of the limb most marked towards the radial border. The lower extremity of the ulna was unusually prominent at the inner border of the forearm, evidently caused by the outward displacement of the hand upon the forearm. Vigorous attempts were made to reduce the deformity, but failed completely. All attempts to produce bony crepitation failed, because, owing to muscular rigidity, reduction could not be effected.

Dissection.—The tendons were first exposed around the wrist-joint. Some effusion of blood was found among the tendon-sheaths, but not in them, although some were unduly filled with clear watery synovia. There then appeared through the tendons an oblique fracture of the lower end of the radius. The fracture was oblique from before backwards and from below upwards and outwards. The fragment was thickest at the root of the styloid process, and so displaced that the process just named pointed backwards and outwards from its usual downward and slightly forward direction. Thus the fragment lay to the posterior and outer surface of the radius at an acute angle and appeared so rotated that its carpal surface looked downwards and backwards. The periosteum on the posterior aspect of the piece detached was much infiltrated with blood, and could easily be removed by the scalpel. The blade of the scalpel could now be inserted into the fissure of the fracture, and the lower fragment, as yet immovable, exposed to view.

Strenuous attempts (extension from the hand and counter-extension by foot against the side of the chest) were repeated to overcome the displacement, but the tendons surrounding the joint became rigid and effectually resisted extension. The tendon of the supinator longus being prominent and appearing directly to retract the fragment was severed, but the displacement continued. Next the flexor carpi radialis and the extensores carpi radiales longior et brevior tendons were divided, and then by bending the hand laterally inwards (that is adducting it on the forearm), the fissure between the upper and lower fragments could be made to gape, and the lower could be nearly replaced in its normal relation to the upper, but it was not till the extensors of the fingers were severed that this could be effected by gentle

traction. Both internal and external lateral ligaments were entire. The loose membranous synovial bag around the lower end of the ulna was thickened by infiltration and patched by ecchymoses ; the lower edge of the pronator quadratus (which corresponded with the chink between the fragments in front) was torn, and a few of its lower fasciculi were softened and blood-stained.

When the soft structures were cleared away the fracture was observed to be more extensive than was before apparent. It was a quarter to half an inch above the carpal extremity, which was completely severed from the shaft of the bone. In general it was transverse from before backwards, but the outer part was very oblique from above downwards and from without inwards and slightly forwards. The lower fragment was found broken into three pieces, of which the middle one was small, and posterior. The two inner ones were in a measure concealed from view before the soft parts were removed. There was no impaction after death, and, in fact, the comminuted state of the lower fragment precluded impaction.

Mr. Couper concludes his remarks as follows:—"This case appears to show that the deformity of Colles's fracture may be entirely due to muscular action, and that the absence of crepitus and irreducibility of the displacement may be due to the same cause, and do not necessarily imply impaction. Mr. Hutchinson assures me that the peculiar deformity of Colles's fracture was entirely absent during life and during paralysis of that side of the body, while I am equally satisfied that during rigor mortis (which was complete) the contour of the wrist exhibited the deformity of Colles's fracture in a marked degree."

My own observations on the mechanism of the muscles which bring about the deformity, when stated before the Hunterian Society during the past winter, brought this case to light ; and it affords remarkable and conclusive proof of the correctness of those views.

The older writers laid great stress on the action of the pronator quadratus.¹ I doubt if it can exert much influence, but the greater part being attached to the upper fragment such force as the damaged little muscle can exert will tend to draw this fragment towards the ulna. Hilton, in a lecture very un-

See Pouteau, '*Œuvres Posthumes*,' 1788, tome ii, p. 255.

worthy of his reputation, speaks of the pronator quadratus acting on the *lower* fragment.¹ This is obviously incorrect.

III. *Fracture of the styloid process of the ulna and rupture of the triangular fibro-cartilage are frequent complications.*

In two out of the three cases that I have dissected I found the styloid process of the ulna detached. In the case dissected nine years after fracture, the styloid process was found united to the end of the ulna by fibrous tissue about two lines in length.²

By pointing out that this process is frequently broken off I hope to correct an inaccuracy which has crept into our text-books, where prominence of the styloid process of the ulna is spoken of instead of prominence of the lower end of the ulna. Thus I find in the article by Flower and Hulke in 'A System of Surgery,'³ "The styloid process of the ulna is thus rendered extremely prominent." In 'Surgery, its Principles and Practice,' by T. Holmes, "A prominence of the styloid process of the ulna."⁴ In 'Science and Art of Surgery,' by J. E. Erichsen, "The styloid process of the ulna projecting sharply under the skin."⁵ In an 'Index of Surgery,' by C. B. Keetley, "Prominence of styloid process of the ulna."⁶ In the 'Science and Practice of Surgery,' by F. J. Gant, "The styloid process of the ulna projecting beneath the integument."⁷

Colles himself does not make this mistake (though he does not appear to have noticed the injury to the styloid process), and Bryant,⁸ who quotes Colles's description, escapes from the

¹ "In the fracture I am now describing the pronator quadratus separates the ends of the fractured radius by drawing the broken lower fragment towards the ulna, but somewhat forwards" ('Guy's Hospital Reports,' 1865, p. 343).

² Fracture of the styloid process has been figured by Malgaigne, and noticed by Croly and Bennett. (See 'Lancet,' June 14th, 1882, p. 1078). Malgaigne's case is copied by Anger, who gives both back and front views and acknowledges it as "Pièce communiquée par Maisonneuve à Malgaigne," 'Anatomie Chirurgicale,' B. Anger, p. 611.

³ 'A System of Surgery,' 3rd ed., vol. iii, p. 966.

⁴ 'Surgery, its Principles and Practice,' 2nd ed., p. 247.

⁵ 'Science and Art of Surgery,' 6th ed., vol. i, p. 325.

⁶ 'Index of Surgery,' p. 126.

⁷ 'Science and Practice of Surgery,' by J. F. Gant, 2nd ed., vol. i, p. 639.

⁸ 'The Practice of Surgery,' 2nd ed., vol. ii, p. 385.

error. "The extremity of the ulna," says Colles, "is seen projecting towards the palm and inner edge of the limb."

Some damage to the ligaments attached to the lower end of the ulna has been generally admitted, and is implied by the following observation of Colles: "If the surgeon proceeds to investigate the nature of this injury, he will find that the end of the ulna admits of being readily moved backwards and forwards." And further on: "The facility with which (as before noticed) the ulna can be moved backwards and forwards does not furnish him with any useful hint." But I do not find mention made of fracture of the styloid process, or of rupture of the triangular fibro-cartilage.

Rupture of the fibro-cartilage I found in two out of three cases, whereas in one neither the cartilage was ruptured nor the process broken. This rupture of the fibro-cartilage does not take place at the base of the styloid process, as might be anticipated, but the base of the cartilage becomes detached from the edge of the radius. I here speak only from my own experience. It is possible that others may find it sometimes detached from the ulna. In the case dissected nine years after fracture there was a free communication between the lower radio-ulnar joint and the wrist-joint.

As regards the frequency of fracture of the ulnar styloid I am able to append some information through the kindness of Professor Gordon, of Belfast, who allowed me, in August last, to examine the unique collection of Colles's fractures in the Queen's College Museum. I found there thirty-four specimens of Colles's fracture, each with the ulna attached. In examining these thirty-four cases for fracture of the ulnar styloid I put two aside as doubtful, because the bones suggested that the absence of the process might be the result of maceration. Of the remaining thirty-two specimens the process was detached in sixteen and present in sixteen. Subsequently I found that four of these specimens had been artificially produced, and of these only one had the styloid process fractured. Reducing the total thus to twenty-eight, there were fifteen specimens in which the styloid process was broken, and thirteen in which it had escaped injury. If to these we add my three cases we get a total of thirty-one, including seventeen in which the process was fractured, and fourteen in which it escaped.

IV. Explanation of the freedom of movement regained after a time, though the deformity remains permanent.

It is within the experience of all that cases of Colles's fracture, in which the deformity has been unreduced and afterwards rendered permanent by the process of repair, regain after a time almost complete mobility at the wrist. I will again quote Colles on this point: "One consolation," he says, "only remains, that the limb will at some future period again enjoy freedom in all its motions and will be completely exempt from pain; the deformity, however, will remain undiminished through life."

It is to be remembered that besides the displacement of the ulna, the articular surface of the radius is permanently reversed from a downward, forward, and inward direction to a downward, backward, and outward direction. It appeared to me, therefore, that either I must find the capsule of the wrist-joint extraordinarily loose or some supplementary movement would be found in the vicinity; for to bend the wrist forward must bring the first row of carpal bones too soon upon the anterior edge of the articular surface of the radius, and the movement would be checked. My dissection gave a complete explanation, inasmuch as it proved that the movements of the wrist-joint were largely supplemented by an abnormal amount of mobility developed in the mid-carpal joint. The capsule of this joint was much more loose than normal, and the ball-and-socket joint formed by the reception of the os magnum and edge of the unciform in the hollow formed by the scaphoid, semilunar, and cuneiform, allowed of great and exceptional freedom of movement.

V. Explanation of the absence of crepitus.

It is certain that not every case of Colles's fracture is impacted, and also certain that where impaction is not present crepitus may still be absent. The absence of crepitus was noted by Pouteau long before Colles: "À l'égard de la crépitation des os," he says, "elle ne peut servir de rien pour recon-

naître une pareille fracture.”¹ Why is it absent? Not because the fibres of the pronator quadratus insert themselves between the fragments. I have particularly looked for this, and have not found it. To obtain crepitus, it is necessary that one fragment should be moved over the other, the rough surfaces grating one against the other as they pass. In Colles's fracture there is a “mouvement de la bascule,” a simile first used by Pouteau¹ to indicate the rotation of the distal fragment on the proximal, but scarcely exact if for see-saw movement, as we understand it, the fulcrum is required to be central. Taken in its wider sense as a hinge-like movement it somewhat aptly expresses what is present in Colles's fracture. This, then, is the explanation of the absence of crepitus. The extensor tendons acting through the posterior annular ligament (aided perhaps by some untorn periosteum) fix the rotated lower fragment upon the posterior edge of the upper so as to form a kind of hinge or pivot, whilst the anterior lips tend rather to gape. Manipulation may cause some to-and-fro movement by opening and shutting the fracture on this pivot, but no true crepitus is obtained till the contracting muscles are so far overcome by extension or chloroform, as to allow one fragment to pass across the face of the other.

VI. Owing to shortening of the radius the head of the ulna may be found below the level of the articular surface of the radius, and the articular surface on the ulna for the sigmoid cavity of the radius may be correspondingly raised.

This point I omitted to search for in the recent cases, and in these, owing to the detachment of the fragments during dissection, it would probably be difficult to verify. In the case of long standing I noted that the head of the ulna projected nearly a quarter of an inch below the level of the articular surface of the radius, and the sigmoid cavity of the radius had made for itself a new surface on the ulna, smooth, and apparently coated with cartilage, as an extension upwards of the normal facet.

The following is a rare case in which the lower fragment was found anterior to the upper :

¹ Op. cit., tome ii, p. 256, pub. 1783.

VII. *Compound fracture of the lower end of the radius, with displacement of the lower fragment forwards and compound dislocation of the head of the ulna.*¹

Fracture of the lower end of the radius obliquely directed from behind forwards and upwards, so that the fractured edge of the lower fragment lies anterior to that of the upper, is very rare,² and has hitherto been believed always to be caused by a fall on the back of the hand.

R. W. Smith gives a case, supposed to be of this kind, complicated with dislocation of the ulna backwards, caused by a fall on the back of the hand. "I cannot speak with accuracy as to the anatomical characters of the injury," he says, "having never had an opportunity of examining after death the skeleton of the forearm in those who had during life met with the accident."³ Gordon had never met with the accident in the living, but mentions that there are two specimens of the fracture in the Queen's College Museum, one of which he figures.⁴ He follows Smith in believing that it must be caused by a fall on the back of the hand.

The following case will show that it may be caused in precisely the same way as an ordinary Colles's fracture, of which it may be considered an accidental variety :

A woman, æt. 56, much addicted to alcohol, was running to get out of the way of a vehicle when she trod upon her dress and fell upon her outstretched palms. She was brought to the surgery on September 29th, 1882, and found to be suffering from a simple Colles's fracture of the right arm and a more serious condition of the left. There was on this side a transverse wound between the flexor carpi radialis and supinator longus caused by the fracture of the radius, and another between the flexor carpi ulnaris and flexor sublimis digitorum caused by a dislocation of the end of the ulna. The fractures were put up in the surgery, the wounds being closed with compound

¹ This case is figured in the edition of 'Bryant's Surgery' just issued. It is there described as an impacted fracture, but this is an error.

² Bennett in examining sixty-two specimens of fracture of the lower end of the radius found only four in which the lower fragment was anterior to the upper ('Brit. Med. Journ.,' vol. i, 1880, p. 759).

³ Op. cit., p. 163.

⁴ Op. cit. p. 30.

tincture of benzoin. Erysipelas with diffuse suppuration along the tendons followed, making it necessary to remove all apparatus from the left arm. She was admitted under Mr. Lucas's care in Patience Ward, and free incisions were made to let out the pus. The forearm, though obscured by the swelling, presented the general appearance of a Colles's fracture. Her temperature rose to 103.8° and she became very delirious. Large portions of skin sloughed, and it was thought advisable on October 17th to amputate the arm.

Examination of the forearm showed suppuration of the lower radio-ulnar and the wrist-joint; and the lower fragment of the radius, which was cut off obliquely from behind upwards and forwards, lay in front of the upper and had perforated the skin.

The articular surface of the radius was rotated and directed downwards, backwards, and outwards, precisely as is found in ordinary cases of Colles's fracture. The projection of the back of the wrist and displacement of the hand corresponded with the ordinary fracture. The direction of the force which caused it being also the same, we must regard this merely as an accidental and rare variety of Colles's fracture. See Plate, page 38, fig. 2.

VIII. *A Description of Colles's fracture.*

Colles's fracture is a fracture of the carpal end of the radius, usually situated from a quarter to one inch¹ above its articular surface, and caused by a fall on the outstretched hand.² The

¹ Colles put the fracture at "about an inch and a half above the carpal extremity of the radius," but, as before remarked, he had not dissected a case, and the appearance of the forearm generally gives one the impression of the fracture being higher than it really is.

² The immediate cause of the fracture at this point has generally been attributed to the direction of the force transmitted through the carpus to the radius when a person falls on the palm. Gordon explains its occurrence as a result of over backward extension of the hand at the wrist-joint, putting the anterior radio-carpal ligaments violently on the stretch, and causing fracture by what he terms "cross-breaking strain." This view he supports by producing the fracture artificially on the dead subject by over-extension ('Fractures of the Lower End of the Radius,' by A. Gordon, 1875). Macleod, without alluding to Gordon's work, explained the fracture in precisely the same way, and supported his remarks by exactly similar experiments four years later, ('Brit. Med. Jour.,' July 12th, 1879).

direction of the fracture is generally either transverse or obliquely directed from below backwards and upwards, and from within outwards and upwards.¹ Not rarely the lower fragment is comminuted,² and in very exceptional cases the fracture may be oblique from behind forwards and upwards.³ Accompanying this fracture there is usually either a fracture of the styloid process of the ulna or a tear of the internal lateral ligament of the wrist-joint, and in addition, frequently a rupture of the triangular fibro-cartilage, and such damage to the lower radio-ulnar ligaments as to allow the end of the ulna to be moved more freely than is normal.⁴

The injury gives rise to three prominences :

1. A dorsal elevation most prominent on the radial side, due to the backward displacement of the articular end of the lower fragment together with the carpus and base of the metacarpus.
2. An anterior rounded prominence extending from the edge of the annular ligament upwards for about three inches, caused by

I find, however, both these observers were preceded by the French. Malgaigne recognises both ways of producing the fracture, and after describing Nélaton's experiments of sawing off the olecranon and striking the bones at the elbow when the palm is flat on the ground he gives the credit to M. Bouchet of showing it may be produced "par une flexion exagérée de la main en avant ou en arrière." "M. Bouchet," he says, "a le premier vérifié ce mécanisme; en essayant d'obtenir des luxations du poignet sur le cadavre, il n'arrivait qu'à produire des fractures de l'extrémité inférieure du radius, quelquefois avec d'autres désordres, et notamment avec fracture simultanée de l'apophyse styloïde du cubitus," Malgaigne '*Traité des Fractures et des Luxations*,' tome i, p. 604, 1847; Bouchet, '*Thèse sur les Luxations du Poignet*,' 1834.

¹ R. W. Smith found eighteen transverse out of twenty (op. cit., p. 156). A. Gordon, on the other hand, found that out of twenty-seven specimens nineteen were oblique from before backwards and eight directly transverse (op. cit., p. 4).

² Pathological specimens show a very large proportion of comminuted fractures, but only the worst cases, such as are caused by falls from a great height, as a rule, yield post-mortem examinations. I extract, in illustration, the following from a lecture by Mr. J. Hutchinson:—"During the last few years at this hospital I believe seven or eight good specimens of recent fracture of the lower part of the radius have been dissected. . . . In all of them the characteristic displacement was present during life. . . . In all the fragments were more or less locked together; in none was there any real impaction, and in every one the lower fragment was comminuted" ('*Brit. Med. Journ.*,' June 30th, 1866).

³ As in a case described in this paper.

⁴ See Colles's description.

the fractured ends (of which the upper is usually somewhat anterior) pressing forward the tendons, together with blood effusion among, and serous effusion into, their sheaths. 3. A prominence on the inner side due to the projection of the lower extremity of the ulna.

There is a depression on the posterior and outer aspect of the forearm, commencing abruptly above the dorsal prominence and fading away on the radial border, due to the displacement of the fractured ends forwards and inwards. Owing to the depression of the radial border of the forearm and the prominence of the lower end of the ulna, the hand presents the appearance of abduction. The movements of pronation and supination are lost. Pain is felt at the seat of fracture and on the inner side of the wrist and hand. Pain in the latter situation is increased by pressure below or on the projecting end of the ulna, and is due to stretching of the dorsal branch of the ulnar nerve.

Examined in the usual way, that is, by attempting to move one fragment on the face of the other, crepitus is rarely obtained. The absence of crepitus is to be explained in two ways :

1. The fracture may be impacted.¹ 2. The muscles acting through the posterior annular ligament may so fix the posterior edges of the fracture that only a slight hinge-like movement can be obtained, which is insufficient to produce crepitus.

On grasping the hand of the patient and making free exten-

¹ Voillemier described four ways in which the fragments might be impacted. 1. Impaction of both walls of the upper fragment into the cancellous tissue of the lower. 2. A similar impaction accompanied with comminution of the lower fragment. 3. Impaction of the posterior edge of the upper fragment into the cancellous tissue of the lower. 4. Reciprocal impaction, the posterior wall of the upper fragment into the cancellous tissue of the lower and the anterior wall of the lower fragment into the cancellous tissue of the upper ('Archives Générales de Médecine,' 1842). He believed that the third form was the most frequent, and surgeons, for the most part, have accepted this opinion as correct. Bennett, however, is of opinion that "in comminuted fractures reciprocal impaction is the most common form of the accident," and that the ratio of comminuted fractures to simple is almost as two to three ('Brit. Med. Journ.,' 1880, vol. i, p. 759). In considering these two points I would remark that evidence of impaction, reciprocal or otherwise, must be accepted with great caution when taken from cases after union is complete; and (as I have before remarked) the relative frequency of comminuted to simple fractures cannot be correctly decided by a collection of pathological specimens, inasmuch as the severity of the accident contributes to the number of specimens obtained.

sion the deformity may be made to disappear, and if at the same time pressure be put on the radius at the seat of fracture a sense of yielding and sometimes crepitus¹ will be felt. On releasing the hand the deformity will recur.²

The chief displacement is of the lower fragment, which becomes rotated on a transverse axis, so that its articular surface instead of looking downwards and slightly forwards is directed downwards and backwards;³ and to a less extent on an antero-posterior axis, so that the articular surface is inclined somewhat outwards instead of inwards. The fractured ends are directed forwards and inwards, the upper fragment being usually somewhat anterior to the lower.

The radius is shortened as a whole and the anterior surface becomes longer than the posterior. The hand follows the shortened radius and is displaced outwards. When the fracture is caused by falls from a height the lower fragment is often comminuted. In this case the most regular fragment is one cut off by a fissure commencing in the ulnar facet, and either breaking out on the dorsum in the groove for the common extensor or reaching to that of the radial extensors.⁴

One cannot fail to be struck with a certain analogy between this fracture and Pott's fracture at the ankle. In both there is fracture with partial dislocation. In the lower extremity the fibula is fractured, the foot displaced outwards, and the inner malleolus fractured or the internal lateral ligament ruptured. In Colles's fracture the radius is fractured, the hand displaced outwards, and the ulnar styloid fractured or its ligaments torn.

¹ Smith, loc. cit.

² Colles, loc. cit.

³ The concavity in front of the lower end of the radius is replaced by a convexity in a bone united in deformity, and the anterior surface is then readily mistaken for the posterior. I showed the lower half of a radius thus deformed to a class of students, and every one mistook the anterior surface for the posterior.

⁴ Packard quoted and verified by Bennett (loc. cit.).

EXPLANATION OF PLATE

Illustrating Mr. Lucas's paper on Colles's Fracture.

FIG. 1. Dissection of wrist after Colles's fracture.

- A. The dorsal branch of the ulnar nerve displaced by the projecting ulnar.**
- B. Bursa over displaced ulna.**
- C. Tendon of extensor carpi ulnaris displaced.**

FIG. 2. Radius from the same case as the dissection, showing the backward direction of the articular surface.

- P. Palmar surface.**
- D. Dorsal surface.**

A

B

C

Fig 1

Fig 2

Hanhart imp

C A S E S
OF
AORTIC ANEURYSM OPENING INTO THE
PULMONARY ARTERY.

BY FREDERICK TAYLOR, M.D.

AMONG the many and various ways in which aortic aneurysms may terminate, there is one of which our knowledge is not very complete, the number of instances on record being comparatively limited. I refer to a rupture into, or communication with, one or other cavity of the heart, or with one of the large vessels immediately adjacent, but I shall confine myself on the present occasion to a consideration of the cases in which the aneurysm opens into the pulmonary artery.

In January, 1868, Dr. Peacock exhibited a case of the kind before the Pathological Society, and he has supplemented the account of it in the nineteenth volume of the 'Transactions' with a table which comprises, including the case exhibited, thirty-three instances of aneurysms opening into the heart, the pulmonary artery, or the vena cava superior. The analysis of the table shows that a communication with the pulmonary artery is the most frequent, and that the next most frequent is that with the superior vena cava; the latter is most often involved by aneurysms of the ascending arch, the former by aneurysms arising immediately at the commencement of the aorta. Besides these thirty-three, Dr. Peacock alludes to six

OF THE SPONTANEOUS ANEURYSM

THESE ANEURYSMS OF THE "PULMONARY" ARE EXISTING IN VARIOUS PLACES AND ARE DESCRIBED BY LAMARCA, making in all 17 cases. THESE CASES ARE VARIOUS IN THE CAVITY OPENED, ARE

1. IN THE CAVITY OPENED INTO THE VENA CAVA SUPERIOR.
2. IN THE CAVITY OPENED INTO THE PULMONARY-VENTRICULAR APERTURE.
3. IN THE CAVITY OPENED INTO THE PULMONARY ARTERY.
4. IN THE CAVITY OPENED INTO THE PULMONARY VEIN.
5. IN THE CAVITY OPENED INTO THE PULMONARY VEIN.
6. IN THE CAVITY OPENED INTO THE PULMONARY VEIN.
7. IN THE CAVITY OPENED INTO THE PULMONARY VEIN.

THESE ANEURYSMS CAUSED THE DESTRUCTION OF LIFE AFTER THE ESTABLISHMENT OF THE ANEURYSM COMMUNICATION, and the physical signs of ANEURYSM WERE RESULT THERE OF, and to these I shall refer later.

THESE ANEURYSMS ARE DESCRIBED IN THE RECORDS AND MUSEUM OF GUY'S HOSPITAL IN WHICH A NUMBER OF ANEURYSMS OPENING INTO THE PULMONARY ARTERY. I HAVE A NUMBER OF CASES RESULT, for while prior to 1840 I HAVE BEEN FROM 1840 TO 1855 INCLUSIVE, I CAN ONLY SAY A FEW CASES HAVE OCCURRED IN THE LAST SIXTEEN YEARS. THE FIRST CASE BEING ONE CASE IN WHICH ANEURYSM IN THE CAVITY OF THE PULMONARY MIGHT BE OPEN TO DOUBT; IN THE SECOND BETWEEN THE TWO VESSELS WAS THINNED DOWN TO A POINT AND IT WAS DR. MARSH'S CONCLUSION, AFTER SEVERAL DISSECTIONS, THAT THE COMMUNICATION ACTUALLY HAD BEEN MADE BY THE PULMONARY AND WAS AN IMMEDIATE CAUSE OF THE CASE. THE SPECIMENS WHICH EXIST IN THE GUY'S HOSPITAL MUSEUM ARE ALL FROM THESE CASES. AT THE PATHOLOGICAL SOCIETY A SMALL NUMBER ONLY HAS BEEN EXHIBITED SINCE DR. MARSH'S CASE IN DR. MARSH'S IN THE SAME YEAR, ONE IN 1841, AND OF THE CASES THAT DIED AT GUY'S HOSPITAL WAS SHOWN IN 1844; AND IN THE SPRING OF 1845 A CASE WAS SHOWN BY DR. S. WEST, ON WHICH OCCASION I ALLUDED TO SOME OF THE OTHER CASES THAT HAD OCCURRED IN RECENT YEARS AT GUY'S HOSPITAL.

IN DESCRIBING THE FEATURES OF THE CASES WHICH I NOW PUBLISH,

OF ANEURYSMS AND ESPECIALLY SPONTANEOUS VARICOSE ANEURYSMS OF THE PULMONARY ARTERY AND VESSELS OF VALVULA WITH CASES," 'Med.-Chir. Trans.' 1840, 1841.

it is interesting first of all to refer back to Mr. Thurnam's paper in the 'Medico-Chirurgical Transactions' already alluded to. Mr. Thurnam carefully discusses as instances of "varicose aneurysm" a number of cases in which the aorta communicated with the *venæ cavæ*, or the cavities of the heart. He defends the application of this surgical term to cases in which an aortic aneurysm communicates with any part of the venous system, whether it be one of the *venæ cavæ*, the pulmonary artery, the right auricle, or the right ventricle, and points out that there are instances in which the lesion has a spontaneous and not a traumatic origin.

He records twelve cases, of which three are instances of communication with the inferior vena cava; the remaining nine are included in Dr. Peacock's table, the pulmonary artery being involved in five cases, and in the four others the superior vena cava, the right auricular appendix, the right auricle and superior cava, and the right ventricle respectively.

From these twelve cases he draws some general inferences, and formulates the points in diagnosis which he thinks may be considered indisputable; and it will be of interest to have them before us.

"*General signs.*—Severe and rapidly advancing anasarca of such portions of the body as are below, or the venous system of which is distal to the varicose orifice. When the varicose aneurysm is between the descending aorta and inferior cava, the legs, scrotum, and lower half of the body; when between the ascending aorta and the superior cava, the arms, face, and upper half of the body; and when between the ascending aorta and one of the right or left cavities of the heart or the pulmonary artery, the whole body is the seat of the dropsical effusion.

"2. Livor of the face particularly, but likewise in a less degree of all such portions of the body as are below the varicose orifice.

"3. A distended and even varicose condition of the subcutaneous veins distal to the orifice.

"4. Dyspnœa, often amounting to orthopnœa and terminating in apnœa.

"5. Cough with expectoration, especially if the sputa be bloody.

"6. A remarkably jerking, and in some cases very feeble, pulse.

"7. Emaciation, debility, loss of muscular power, deficient animal heat and sensorial disturbance, may be looked upon as somewhat less frequent and certain signs.

"8. *Physical signs.*—A superficial, harsh, and peculiarly intense sawing or blowing sound, accompanied by an equally marked and purring tremor, heard over the varicose orifice and in the current of the circulation beyond it; this sound is continuous, but is loudest during the systole, less loud during the diastole, and still less so during the interval."¹

Dr. Peacock in his summary² speaks with much less confidence of the diagnosis. "In several cases in which marked evidences of venous derangements of the upper parts of the body suddenly occurred the nature of the affection has been correctly diagnosed during life, but where the openings are into the pulmonary artery, right ventricle, or left auricle, the diagnosis will generally be attended with great difficulty and uncertainty."

The caution is, I think, amply justified by the cases which are here published. In Mr. Thurnam's group there was an unusual preponderance of cases affecting the veins, whereas in Dr. Peacock's list the ruptures into the pulmonary artery are shown to form a large majority. The symptoms tabulated by Thurnam are those of venous obstruction, and the nearer the obstruction to the heart the closer the resemblance of the symptoms to those of the commonest form of venous obstruction, that which arises from cardiac valvular disease. There, indeed, is the difficulty of diagnosis, at least in the cases now under consideration, namely, those affecting the pulmonary artery; they may be so readily confounded with cases of valvular heart disease. The thrills and murmurs which have so much value for the surgeon in arterial disease in the limbs, give much less certain indications when the disease is situated in or about the heart.

The diagnosis mostly requires a double effort. The recognition of aneurysm at the base of the heart is by no means so simple as it is in the course of a limb, but aneurysm may be diagnosed without a suspicion of a communication with the

¹ Loc. cit., pp. 373, 374.

² Loc. cit., p. 124.

pulmonary artery. On the other hand, if good evidence could be afforded of a communication between the arterial and venous systems about the heart, there would be a strong inference in favour of aortic aneurysm in a case where the symptoms were developed with advancing age, and a patent ductus arteriosus was therefore out of the question. It is the evidence of the communication that is desired first of all, and the question naturally suggests itself whether there is any auscultatory sign that is constantly present. Twenty-six cases are before me, where an aneurysm communicated with the pulmonary artery, namely, fifteen by Peacock, one by Murchison, one by Cayley, one by S. West, one my own case in the '*Pathological Transactions*,' and seven in the present paper. In seven of these death occurred too promptly for record of the physical signs to be made.

The remaining nineteen cases are tabulated below, forming three groups, namely, seven cases with a systolic murmur only, six with a continuous murmur, covering both sounds or running from one period to the other of the heart's rhythm, and six with a murmur described as double, or to-and-fro. Some kind of thrill was present in four of the first group, in three of the second, and two of the third group, nine in all. It must not be forgotten that these observations have but little value unless there is evidence that at the time of the observation the communication did actually exist, as of course the very presence of an aneurysm, with the various changes which it may effect about the heart, might have been sufficient to produce the murmurs heard. Dr. Peacock states that a majority of his cases of rupture into the pulmonary artery died quickly after the accident, but in twelve of the nineteen cases here grouped together, there appears to be evidence of a communication of some standing, either in the thick rounded edges of the aperture (Dr. Peacock's Cases 9, 10, and 27, Dr. Murchison's case, and Cases 2, 4, 5, and 7 here published), or in the condition of the pulmonary artery show that a current of blood from the aorta impinged upon it (Cases 5 and 6 in this paper), or in the presence of vegetations which might at least take some days to form (my case, R. B—, in the '*Path. Trans.*,' vol. xxv); or, finally, in the history of a sudden aggravation of symptoms (Dr. Peacock's Case 20, and Dr. S.

West's case). The aperture was inferred to be quite recent in Case 3 of the present paper, and in Dr. Peacock's Case 38, in which it had very thin, smooth, membranous edges. In the case he himself recorded in the 'Pathological Transactions' (Case 32), Dr. Peacock thought it "most probable that the communication between the aneurysmal sac and the pulmonary artery and right ventricle only occurred shortly before death, and was in no degree connected with the auscultatory signs which had been observed during life." In Dr. Cayley's case the margin of the opening was thin and smooth, but in the history there is no recent occurrence of sudden aggravation of symptoms; and Dr. Cayley thought the case was so complicated that little value could be attached to the ascription of any particular symptoms to the communication between the two vessels. The data for forming an opinion in Dr. Peacock's cases 17, 25, and 26 are scarcely sufficient.

Cases with a Systolic Murmur.

Case.	Murmur.	Thrill.
From Dr. Peacock's table, Case 9 (Smith's)	Bruit with systole	Intense frémissement over whole præcordia.
Case 20 (Ogle's)	Systolic murmur in præcordia loudest at left side of sternum, opposite third cartilage; clear diastolic sound on right, but none on left of sternum	Tremor with the systole.
Case 26 (Pierreson's)	Souffle with the first sound	Purring tremor.
Dr. Murchison's Case, 'Path. Trans.,' vol. xix, p. 191	Over the whole of the cardiac region was a prolonged systolic bellows-murmur, loudest at the apex, but also very loud at the base and at mid-sternum, where it was louder than over the space intervening between this and the apex; it was also heard at the back of the chest, but was not propagated upwards along the great vessels.	
Cases in the present paper. Case 2 (George P—)	A systolic murmur is heard at the upper border of the second costal cartilage, as well as down the sternum. There is no diastolic murmur	Systolic thrill in the second left intercostal space.

Case.	Murmur.	Thrill.
Case 5 (Charles P—)	A murmur was heard over the sternum, and widely on both sides of it, but its greatest intensity was to the left of the sternum, near the third and fourth cartilages, and corresponded to neither the aortic, pulmonary, nor tricuspid orifices. There was no diastolic murmur.	
Case 7 (Joseph E—)	Systolic bruit at apex (?)	

Cases with a Continuous Murmur covering both Sounds.

Dr. Peacock's Cases, Case 10 (Munro's)	The first sound was accompanied by a loud blowing murmur, most distinct at the middle of the sternum, but audible over the whole fore part of the chest, and over the back on both sides of the spine. The second sound was short and much obscured by the first. ("Hence," observes Dr. Hope, "it appears as a continuous murmur, extended from the first over the second sound.")	
Case 17 (Turnbull's)	Loud and harsh double murmur, continuous, but most intense with the systole.	Distinct purring sensation at base.
Case 25 (Bennett's)	At the junction of left third costal cartilage with sternum, first sound prolonged and blowing, second short, abrupt, and rasping; over manubrium, rough continuous blowing murmur, occupying both sounds. Soft blowing systolic murmur and natural second sound at apex.	
Case 33 (Roberts')	A superficial, loud, harsh double murmur, or a single murmur consisting of two parts and covering both sounds, heard most intensely at the upper level of cardiac dulness, and between sternum and nipple; propagated to the vessels in the neck and heard much less intensely at the apex	Intense vibratile thrill.
Dr. West's Case, 'Path Trans.,' 1883, vol. xxxiv	A double cardiac murmur propagated to the left side; began at the end of diastole and went through the whole of systole	A coarse thrill was felt in the third and fourth spaces to the left of the sternum.
Case 6 in the present paper (Thomas F—)	At the base of the heart, to the left of the sternum, was a peculiarly roaring, musical bruit, so prolonged as to occupy the entire systole and run one systole into another. No diastolic murmur	No thrill.

Cases with a Double Murmur.

Case.	Murmur.	Thrill.
From Dr. Peacock's table. Case 27 (Wade's)	Cardiac sound was heard over the cartilage of the fourth rib, replaced by two murmurs; that with the second sound occupied the whole period of silence; a loud bellows-murmur heard also with the first sound. At apex a single systolic murmur	Purring tremor at the fourth cartilage.
Case 32 (Peacock's)	At base loud systolic murmur, followed by loud, ringing, diastolic sound, and that by a slight murmur. At apex only the systolic murmur.	
Dr. Taylor's Case. R. B—, 'Path. Trans.,' 1874, vol. xxv Dr. Cayley's Case, 'Path. Trans.,' 1870, vol. xxi	Double murmur at the base, the systolic louder. Systolic also at the apex. A double bellows-murmur at the second costal cartilage and along the left edge of the sternum.	
Cases in the present paper. Case 3 (William H—)	Loud to-and-fro murmur is heard down the sternum, near the ensiform cartilage, and over the third right costal cartilage. At the apex there is a soft blowing systolic murmur.	Marked thrill in the second and third left inter-costal spaces.
Case 4 (Alfred W—)	A very loud blowing systolic murmur and a fainter diastolic. The systolic is heard all over the front of the chest, distinctly in the left axilla and less distinctly behind	

The cases in which the murmur could be considered peculiar, or markedly different from what one may hear in ordinary aortic cardiac cases, are Cases 10, 17, 25, 27, and 33 by Dr. Peacock, Dr. S. West's case, and Case 6 in the present paper.

In most of these I have shown there was evidence that the communication was of some standing, and may therefore have given rise to the auscultatory signs recorded. All but one fall into the second group, in which neither systole nor diastole were entirely free from murmur, but the murmur, though in that sense double, was rather of a continuous than a to-and-fro character. In Dr. West's case the description differs from the others, in that he regarded the murmur as coming

at the end of diastole and running through systole, whereas in others it seems to have begun with systole and continued into diastole.

In Case 6 it is expressly stated by Dr. Goodhart, whose case it was, that the murmur was systolic, "so prolonged as to run one systole into another," and yet there was "no diastolic" murmur. This no doubt means that the sound covering the period of dilatation was continuous with, and produced by, the same contraction or moving force as that which produced the systolic portion proper.

From Dr. Wade's account of his own case (*Med.-Chir. Transactions*, vol. xliv) it appears that the murmur was distinctly double, but the diastolic portion was hissing and peculiarly prolonged; it was accompanied by purring tremor.

As to the situation of the murmur it was in all cases near the base of the heart, and in some at least over the pulmonary area. Thus in the various cases it was "at the middle of the sternum" (Dr. Peacock's Case 10); over the manubrium "at the upper level of cardiac dulness, and between the sternum and nipple" (Dr. Peacock's Case 33); at the base of the heart to the left of the sternum (Thomas F—, Case 6); "propagated to the left side" (Dr. West's case). In only two of these six was a thrill present.

Clinical experience then plainly shows that communication may take place between aortic aneurysms and the pulmonary artery without any characteristic physical sign; and that only in a small proportion of cases will the physical signs suggest an exceptional state of things. We may still, however, ask whether in this minority the physical signs correspond to the morbid conditions, and what it is in the majority of cases which prevents these physical signs from occurring? If for a moment we regard these cases as ordinary varicose aneurysms, and ask what are the symptoms of the same thing in the course of a limb, we find writers on surgery speaking of the buzzing thrill, or vibratory thrill, or burring bruit, as characteristic of varicose aneurysm, which is further distinguished by its continuous murmur from a simple aneurysm.

Thurnam¹ assumes the same kind of murmur from all communications of an aneurysm with the cavities of the heart,

¹ *Loc. cit.*, p. 372.

or vessels near it, and gives the following detailed description :

“As a consequence of the superior force of the left ventricle, the arterial blood is doubtless propelled through the varicose orifice, and so produces the murmur. During the systole of the heart the current through the orifice is the strongest, and the sound consequently is then the loudest. During the diastole, in consequence of the elastic reaction of the arterial system on its contained blood, a less powerful current is propelled through the opening, and at that time a somewhat weaker murmur is heard. This reaction of the arteries, however, is in operation not only during the diastole, but also during the interval, and, in fact, until it is overcome by the succeeding ventricular systole ; consequently, though the current is stronger at the commencement of this reaction, and synchronously with the diastole, yet it is also continued during the interval. Hence the murmur is a continuous one ; it being present, though much weaker, during the interval between the diastole and the succeeding systole. The same circumstances which produce the murmur of course occasion the purring tremor.”

The cases here published and referred to show that this account, though no doubt a correct explanation of the murmur in some cases, is certainly not true for all cases of communication between an aortic aneurysm and the pulmonary artery ; indeed, as a record of observation, it is scarcely justified by Thurnam's own cases, as applying to those in which the communication most closely involved the heart. For out of his twelve cases, six died so quickly as to leave us without record even of the existence of a murmur, and only in three is the description sufficiently detailed to correspond obviously with the above account. It is especially interesting, with reference to the group now under consideration, that these six cases of rapid death without any record of murmur include four out of his five cases of rupture into the pulmonary artery.

There must be other elements or factors in the majority of these cases which interfere with the simple conditions which are required for the continuous murmur described, and which exist perhaps always in the varicose aneurysms of the limbs.

It would at first sight appear that the requirement for the production of the continuous murmur is the transference of fluid from one vessel to the other with a sufficient velocity, that is, proportionate to the size of the aperture, during both systole and diastole. This passage of blood by which the vibrations are brought about, must depend on a difference of pressures on the two sides of the communication. The pressure on the side of the aorta is mostly, if not always, greater than that on the side of the veins and pulmonary artery; certainly during systole, less certainly during diastole; so that when a communication is opened between them there should be a passage of blood from the aorta to the pulmonary artery both during systole and during diastole, though the pressure might fall so low towards the end of diastole as to be insufficient then for the production of an audible sound. Such a murmur would be continuous, seeing that it is due to a single uninterrupted *veine fluide* or eddy-formation in the cavity of the pulmonary artery, and not to two separate jets such as in typical aortic valve disease form in the aorta and left ventricle alternately, and give rise to the characteristic to-and-fro murmurs.

The conditions interfering with this simple continuous murmur may depend on the influence the aortic valves, the left ventricle, and the right ventricle have upon the blood pressure about the orifice of communication. The difference of pressures required for the transference of the blood may be lessened by an increase on the side of the pulmonary artery, or a decrease on the side of the aorta; and in reference to the latter it must be remembered we are not dealing with a healthy aorta but with one the subject of aneurysm. Further, there may be accompanying aortic regurgitation or dilatation and weakened contraction of the left ventricle.

Though I cannot refer to any authoritative statement to this effect, there can, I suppose, be no doubt that the pressure in an aneurysm is less than that in the section of the vessel from which it springs, and this would lead to an approximation of the pressures on the two sides of the aperture.

Though aortic regurgitation is a condition one would expect rather often, it is remarkable in what a large proportion of the twenty-six cases under consideration the aortic valves

were healthy or not largely diseased. Of the nineteen cases in which murmurs were noted bearing on this question, eight had healthy aortic valves, in six they were as follows:—“Slightly thickened, but competent;” “stretched and thickened but practically workable;” “a little thick, practically healthy;” “possibly good;” “aortic orifice somewhat large, but valves closed it nearly, if not perfectly;” “though thickened, did not allow of the regurgitation of water.” In another case they were described as thickened, in another they were bad, but appeared to hold water post-mortem; in another they were much diseased.

In the remaining five patients their condition is not recorded. Some of these, who had valves of only doubtful excellence, appear to have had splashing pulses, and thus possibly the number of cases with bad aortic valves may be really larger than seems at first to be the case.¹

The condition of the pulse during life may also supply evidence of regurgitation, and we find six cases are recorded to have had splashing or collapsing pulses, of which four had double murmurs, but two only a single systolic murmur. So that there is still a little want of correspondence between the pulse, the murmur, and the condition of the valves post-mortem; but on the whole it seems probable that the condition of the valves may sometimes be antagonistic to the development of this particular murmur by diminishing the blood-pressure on the side of the aorta.

Another circumstance which may interfere with the production of a bruit is worthy of attention. The rupture of an aneurysm into the pulmonary artery often does not take place until the aneurysm has for some time pressed upon, and actually bulged into, the cavity of the pulmonary artery. And this pressure alone may suffice to induce very serious symptoms, and even the fatal termination. The pulmonary sigmoid valves may become adherent to that wall of the vessel which is being pushed in by the aneurysm, and it is clear that a considerable degree of narrowing may result. The bulging would thus come into relatively close contact with the opposite

¹ It is to be regretted that sphygmographic tracings are not more often available, and I must plead guilty to want of care in this respect in the few cases I have had personally under my own observation.

pulmonary wall, and if the rupture takes place at this most prominent part, it appears to me that the conditions for the production of a murmur, especially during diastole, may be very much interfered with.

It remains to consider whether cases of patent ductus arteriosus give any assistance to us in this matter; but the information with reference to such cases is itself too scanty to be of much value.

The number on record is but small, they have been diagnosed with difficulty, and there has been no constancy about the murmurs audible. Readers of these reports may remember that the late Dr. Hilton Fagge published the complete history of a case of patent ductus arteriosus.¹ There were two murmurs, a systolic, and a peculiar wavy diastolic murmur, partly musical, and at one spot not directly continuous with the second sound. The systolic and diastolic sounds were apparently separated by an interval, even where the diastolic murmur was continued on from the second sound; and it is the diastolic portion that was regarded as peculiarly the result of the abnormal communication.

Extreme slowness of the heart's beat facilitated the observation of the sounds. In his comments Dr. Fagge contrasted his case with that of Dr. Wade already alluded to, and pointed out the difference in the presence of an aneurysm, and in the direction of the blood current. With reference to this last point it may be observed that in all but three of the twenty-six cases of aneurysm here discussed, the communication was with the trunk of the pulmonary artery and not at the site of the ductus arteriosus. The three exceptions are, Case 7 of Dr. Peacock's list, in which the left main branch was opened, death took place rapidly, and no physical signs were observed. The case of George P— (Case 2), in which the right main branch was opened, and a systolic murmur only was heard, and that of James G— (Case 1), in which the aneurysm arose from the aorta opposite the left subclavian artery, and therefore apparently communicated with the pulmonary artery immediately adjacent. No murmur was heard in this case, the rupture only just preceding death.

Dr. Fagge referred to a case by Kaulich in which a murmur

¹ 'Guy's Hosp. Rep.,' vol. xviii, 3rd ser., p. 23.

somewhat similar to that in his own case was heard, to one recorded by Dr. Babington, and to others, where the signs were of no diagnostic value. In a recent work on Diseases of the Heart¹ M. Paul alludes to six cases of patent ductus arteriosus, including Dr. Babington's. Of these he says: "In two cases there were no murmurs; in another case, that of Sanders, there was only a systolic murmur; in the cases of Almagro, and of M. Bernutz, there were two murmurs, but it must not be forgotten that in these two cases the aorta was constricted."

Recently, Dr. Foulis has published a very interesting case of patent ductus arteriosus, upon which a discussion took place at the Medico-Chirurgical Society of Edinburgh.² The physical signs were marked systolic pulsation in the second left intercostal space, a loud blowing systolic murmur with a decided thrill in it, and a softer, shorter diastolic murmur. These were heard over the site of pulsation, also very distinctly over the aortic area, and again, but not so loudly, at the apex of the heart. At the post-mortem examination the ductus arteriosus was patent, the size of a goose quill, but there was a saccular aneurysm of the pulmonary artery, and such extensive disease of the pulmonary valves that the share of the abnormal communication in the production of the sounds must be very difficult correctly to estimate. Dr. Foulis attributed the systolic murmur to pulmonary valve obstruction together with escape of blood *from* the pulmonary artery *into* the aorta; the diastolic murmur mainly, if not entirely to reflux from the aorta into the pulmonary artery. But this view was not accepted by all who spoke at the debate.

One great difficulty attends this subject, the want of a method of graphically recording the acoustic phenomena of disease, as verbal descriptions often entirely fail to convey any idea of differences which are appreciable to the ear.

If satisfactory auscultatory signs in the heart and great vessels are often wanting when a communication exists between an aneurysm and the pulmonary artery, are there any other

¹ 'Diagnostic et Traitement des Maladies du Cœur.' Par Coustantin Paul. Paris, 1883.

² 'Edinburgh Medical Journal,' July, 1884, pp. 17 and 55.

means by which the condition can be inferred? Dr. Wade, in his oft quoted case, laid stress upon the freedom of the lungs from the results of mechanical congestion, while the liver and other parts feeding the venæ cavæ were engorged, as indicating that the backward pressure was exerted immediately from left to right ventricle, instead of mediately through the pulmonary circulation. But however useful as a sign this freedom of the lungs may be when it is present, the condition of these organs cannot be depended upon, as may be seen by the seven cases at the end of this paper. If we exclude the two cases, in which the rupture only immediately preceded death (Cases 1 and 3), we find that in three of the other five there was induration similar to or identical with that of heart disease, in another case the lung was hepatised, in another both lungs were œdematous, and three out of the five had fluid in the pleuræ. This certainly shows that in a large number of cases conditions are present which do lead to congestion and œdema of the lungs, and to pleural effusion, as well as to obstruction in the systemic venous circulation. One will at least beware of excluding from the diagnosis an aneurysm rupturing into the pulmonary artery, because the lungs are engorged.

If the pulmonary circulation does not help us very much, can we learn anything from the systemic circulation? Dr. Goodhart, in Case 6, was led by the lividity and general anasarca of the patient to diagnose a communication between the pulmonary artery and the aorta; and this, indeed, proved correct. On the other hand, Dr. Murchison, in commenting upon his case, called attention to the "remarkable anæmia," and stated he had seen anæmia also in a similar case. In Dr. Cayley's case there was marked anæmia, but at the same time great lividity. From the other cases here published no definite conclusion can be drawn. No special mention is made of the cyanosis or anæmia in Cases 2, 4, and 5; in Case 3 the face was dusky, with congested lips, and shortly before death the face became very much congested. Case 7, however, was very pale during the last few days of life. Case 1 was much emaciated, no physical signs of cardiac disease were observed, and the rupture is inferred to have taken place just before death. Anæmia could reasonably be explained by the

escape of blood through the aneurysm into the pulmonary artery, and the consequent diminution of the quantity supplied to the systemic arteries; but anæmia occurs in aortic regurgitation, and this condition would have to be excluded before one could attach great importance to anæmia as a diagnostic sign.

On the other hand, cyanosis seems to me still less reliable as a sign of a communication between the vessels of this particular kind. The mixture of the two currents of blood is not a satisfactory explanation of the cyanosis in congenital heart disease, and in these cases it is much less so, since the arterial blood flows into the venous, and not the venous into the arterial; whereas the pulmonary obstruction, which is the main cause of cyanosis in congenital disease, may be brought about in these cases quite independently of an opening between the two great vessels. As I have before remarked, the aneurysm frequently bulges into the pulmonary artery, and may cause dilatation of the right heart, engorgement of the liver, albuminuria, general dropsy, and death, without any communication at all taking place.

CASE 1. *Vomiting; emaciation; absence of physical signs; sudden severe cardiac pain; death. Small aneurysm of the aorta apparently just opened into the pulmonary artery.*

James G—, æt. 30, was admitted under Dr. Owen Rees, June 28rd, 1868. He was a seaman, and had never had any serious disease until twelve months ago when he could not retain food. It returned ten or fifteen minutes after being taken. He has just returned from a long voyage, and has been badly fed, so that he has a starved appearance.

On admission, much emaciated; stomach distended; no tumour felt anywhere; liver and other abdominal organs appear healthy. The thoracic organs are healthy. He has no pain. Constant vomiting was the chief symptom he suffered from, but he kept about the ward in a languid, melancholy way. In the night of June 30, the resident medical officer was called to him and found him in great agony, always referring pain to the stomach and heart. He was ordered

one third of a grain of opium, and poultices were applied to the chest. He obtained some relief, but at 10 a.m. on July 1st he was very collapsed, with severe pain in the stomach, and a scarcely perceptible pulse. He died two hours later.

Dr. Moxon, who made the post-mortem examination, says :

“ The impression on the minds of those who saw him was of some obscure organic change, melasma suprarenale, phthisis, or organic stomach disease.

“ He was five feet nine, very spare but well shaped, and of dark olive-brown complexion with woolly hair, aquiline flat features, something between African and North-American Indian in cast. The forehead retracted, the cranium sunk ; no sores nor scars.

“ Each pleura contained a pint of limpid, slightly yellowish serum, the membrane being quite healthy in appearance. The left lung was loaded with serum, being highly œdematous. They were at the same time of tough consistency ; I thought rather more than the average, so that the state approached that called at Guy’s ‘ heart lung.’ No œdema of larynx.

“ *Heart* weighed 10 ounces ; its size was rather remarkable, because it was as large as usual when the body was much wasted. The form was remarkably rounded, and this proved to be due to the quantity of contents in the flaccid left side. A little gelatinous death-bed clot in the right ventricle, and a little black clot in the right auricle. The pulmonary artery was opened into by the gentlest thrust from a probe in ascertaining the relations of a pouch from the aorta which proved to be a dilated ductus arteriosus.

“ The contents of the left ventricle were about five or six ounces of black gelatinous clot, in the auricle about two ounces, in all a very remarkable quantity which distended and rounded the left heart. The muscle in the columns of the left ventricle was striated across in a manner like ‘ tabby ’ markings. There was obvious fatty degeneration of the substance of the auricle. The right heart appeared not to share in this.

“ The great vessels arose in the usual way, but opposite the left subclavian was such a pouch as would admit the tip of one’s index finger. This ran on into contact with the pulmonary artery, and thinned the coats of this down very

remarkably, so that a very slight touch broke down the partition and brought the pulmonary artery and aorta into most free communication. It is very unfortunate that this probing was done without previous inspection of the pulmonary artery. The symptoms of the fatal seizure might have been due to the rupture of the aneurysm. I found after passing the probe very gently that it went straight into the pulmonary artery. What I first put in was the scissors' point I was using, but I put it in very gently, and I certainly did not feel any resistance or use any force, yet when the blunt probe was used it went straight on into the pulmonary artery. Now, when we take into consideration the remarkable state of the left heart, so exceedingly full of blood-clot, it seems very likely that the rupture of the aneurysm preceded and caused his death. The same conclusion is further borne out by the œdematous state of the lungs, and the liquid in the thoracic cavities. Under all the circumstances I do not doubt that the bursting of the aneurysm, 'varicose aneurysm' was the real cause of his death. It accounts for the seizure on the night before his death being sudden and attended with pain at the heart.

"*Kidneys* had all the characters of typical and healthy organs.

"*Stomach* showed a remarkable condition of mixed ulceration and scarring quite unlike ordinary ulceration."

CASE 2. Dyspnœa and cough for eight weeks ; anasarca three weeks ; systolic murmur in second left space ; albuminuria ; hæmoptysis ; death. Aneurysm opening into pulmonary artery ; both ventricles hypertrophied ; aortic valves diseased.

(Reported by Mr. W. H. COATES.)

George P—, æt. 45, was admitted under Dr. Taylor's care on March 4th, 1874. He had been subject to what he called rheumatic gout in the ankles and elbows two or three times in the year. He has drunk three or four pints of beer daily, and spirits occasionally. The present illness came on suddenly eight weeks ago with "fluttering" in the left hypochondriac region, and this necessitated his giving up his work.

He has been troubled with cough from the commencement of the present attack, but it has been less troublesome during the last fortnight. He has also had pain in the epigastric and left hypochondriac regions after taking food. His breathing has been so distressed that he has found it impossible to lie down. The first three weeks he spat a little blood, now he expectorates frothy mucus without much difficulty. Three weeks ago his feet began to swell, and the swelling has gradually extended up his legs.

Present condition.—Face and lips pale; slight flush on the cheeks. He lies in bed with back and shoulders supported, but his most comfortable position is that in which his body is inclined a little forward and is supported upon the arms. The respiration is wheezing, more abdominal than thoracic; some inspiratory dyspnoea. The chest is resonant on both sides as far as the fourth interspace, and dull below that point. Very loud dry sounds are heard over the right lung, with cooing expiration. The sounds are similar but less loud over the left lung. The heart's apex beats one inch below and two inches external to the nipple. Præcordial dulness is much increased. A systolic murmur is heard at the upper border of the second costal cartilage, as well as down the sternum; there is no diastolic murmur. The urine has a sp. gr. of 1020, and contains albumen. The ankles are swollen.

He slept scarcely at all the night after admission.

On the 6th March he had passed during the preceding twenty-four hours 11 ounces of high-coloured urine with a sp. gr. of 1038, and containing a trace of albumen. His bowels had been opened a little in the night. The expectoration was frothy and tinged with blood. The tongue was moist and thinly covered with brown fur. Pulse 104.

March 7th.—A systolic thrill can be felt in the second left intercostal space.

9th.—His bowels have been freely open by aperients, and the pain in the epigastric region has been relieved, so that he slept seven hours last night. This morning he breathes with greater freedom, and feels altogether more comfortable. His legs are much less swollen.

10th.—Sputum tinged with blood. Breathing easier. Urine, 14 ounces, sp. gr. 1008, contains albumen.

12th.—Last night about ten o'clock he began to spit blood. This has continued all through the night and up to the present time, 10.30 a.m. The blood is bright red, frothy, and has a sour smell. This morning he is restless and depressed; his face is flushed, and the cough troublesome. He had another aperient yesterday, and his bowels have acted three times. He died in the course of the day.

Post-mortem (by Dr. Goodhart).—Both pleuræ contained a considerable quantity of fluid, the right side more than the left, about $1\frac{1}{2}$ pints. Very recent pleurisy over the posterior part of the right lung. The right lung presented in its upper two-thirds a condition of consolidation, at one part like that of ordinary lobar pneumonia, at another like the solid induration found in advanced mitral disease. It was, however, everywhere friable, giving way readily under the finger. The lower part of the lung was airless from pressure and sank in water. The left lung was rather dry and shrunken, but a fair quantity of blood flowed from its vessels on section. The lung was not emphysematous, nor did the bronchial tubes contain any pus. The larger tubes contained a little red mucus, and were perhaps a little dilated. The branches of the pulmonary artery throughout the lung were healthy.

The heart weighed $28\frac{1}{2}$ ounces. The right side shared the apex, and had a large surface area in front, although the left side was also considerably hypertrophied. The pericardium contained about 3 ounces of fluid. On opening it, the intra-pericardial part of the arch was seen to be very large, from above downwards somewhat, but chiefly in a lateral direction, so much so that instead of the pulmonary artery being the prominent vessel, the aorta monopolised attention, overlapping as it did, and pushing somewhat to the left, the main pulmonary trunk. The aorta had formed filamentous adhesions to the pericardium at this part. The vessels of the left side were tested without cutting the heart away from the lungs, and the aortic valves allowed water to run through them leisurely; the mitral appeared to resist the back flow of water fairly well. The aortics, however, were not worse in allowing water to run into the ventricle than many competent valves tested in the same way, and looked at from above they appeared to be good. The aorta was next slit up, and a large

aneurysmal sac was exposed taking up the whole circumference of the vessel and extending from the valve to the innominate aperture above. The arterial coat was not healthy at any part of its surface, and had an irregular aspect from plates of atheroma of various shapes and sizes. Very little calcareous matter was present; the anterior wall of the sac, an inch or so above the valves, had a small secondary pouch projecting from it forwards into the pericardium; this contained no clot, and its walls were very thin. It would probably have burst before long into the pericardial cavity. To the left side of the posterior valve again, an inch or so above it, an opening from the aorta into the pulmonary artery going to the right lung had occurred; the aperture had a nearly transverse direction in relation to the circumference of the artery, and the coats of the aortic and pulmonary artery at its edge were represented by merely a thin membrane. The edges of this opening were rounded as if it had been formed some time. An ounce or so of recent post-mortem clot lay loose in the cavity of the aneurysm, but there was nowhere any laminated or ante-mortem coagulum. Between the left carotid and subclavian vessels the aorta resumed its proper size again rather suddenly, so as to make a distinct annular mouth to the sac. It dilated, however, immediately afterwards into a second small aneurysmal enlargement of the tube for an inch or so, and then again contracting to its proper size remained normal, except that it was somewhat extensively atheromatous. The aortic valves were bad; the posterior valve had two thirds of its meeting line to the left converted into a rounded cord, and one of the anterior valves was very thick, so that as they lay open it would seem that a good deal of regurgitation had existed. The muscular substance of the right side was at least twice its proper thickness and very tough. The endocardium of the left ventricle though generally healthy had just below the aortic valves a white thickened patch arranged in a trabecular manner, the thickening having taken place in certain lines. No cause for friction could be found to account for this patch. The muscle of the left ventricle was good. No fibrous patches.

Extensive atheroma of abdominal aorta. *Stomach* healthy. *Liver* 65 oz., tough and nutmegged. *Spleen* 7 oz., very firm.

Kidneys 21 oz., very hard and coarse, but not diseased. Gout in the great toe-joints.

CASE 3. *Ill two years with dyspnœa, cough, cardiac pain; anasarca three weeks; double murmur, collapsing pulse; rather suddenly worse; aneurysm opening into pulmonary artery by recent rupture.*

(Reported by Mr. G. H. WEST JONES.)

William H—, æt. 44, was admitted under Dr. Habershon's care on January 23rd, 1878. He was in the marine service twelve years, and left it in 1866; since then he has been working as a labourer at the Royal Arsenal, Woolwich. He was six years in China, and while there had ague. In 1855 he had chancres, but gives no history of sorethroat, or other secondary symptoms.

Two years ago he was working on the marshes, when he had shivering; he attended a doctor, but kept on working. He has gradually got worse, having severe pain in the region of the heart, a troublesome cough, and gradually increasing shortness of breath. A fortnight ago his legs began to swell, and he has been laid up ever since.

Present condition.—Well nourished, dark complexion, dusky face, with very congested lips. He is easiest when lying on his right side. There is œdema of the feet, legs, and chest. The abdomen is rather tumid, and the liver dulness extends two inches below the ribs, but there is no ascites. There is considerable dyspnœa. Respiration is chiefly abdominal.

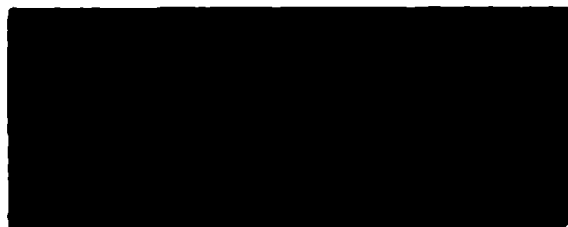
The chest is well formed, but the movement is impaired, and more so on the right side than on the left. Vocal fremitus is not well marked. The breath-sounds are very harsh at the right apex, and at both bases there is slight crepitation.

Præcordial dulness is increased in a downward direction. The impulse is very forcible, it can be seen between the sixth and seventh ribs, and can be felt as far out as the axilla. There is also well-marked epigastric pulsation. A loud to-and-fro murmur is heard down the sternum, near the ensiform cartilage, and over the third right costal cartilage. At the apex there is a soft blowing systolic murmur.

The radial pulse has a well-marked water-hammer character, indicative of aortic disease; and the brachials are very visible at the bend of the elbow.

Urine, sp. gr. 1022, deposits urates, contains no blood nor sugar nor casts, but is albuminous.

Till January 28th he had slightly improved, but his cough was then more troublesome, and he did not seem so well.



Pulse tracing, February 4th; pressure, 5 oz.

During the next week, cough and dyspnoea continued, and the swelling of the legs increased.

On February 6th he became very much worse, the face very much congested, the pulse hardly perceptible. Venesection was attempted, but no blood flowed, and the patient died.

Post-mortem.—Red induration of base of right lung.

Pericardium adherent throughout.

Heart weighed 28 oz.; all parts enlarged, the cavities all dilated, especially the two ventricles. The mitral valves stretched, and admitting four fingers easily. The auricular surface of the ventricular flap, nodulated from old thickening at its free edge; the ventricular aspect not showing any disease. The posterior aortic cusp rather thick at its edge, and the valves did not now meet well, but they were not really bad valves. The aorta immediately above the valves was extremely diseased and dilated into a large rounded sac about the size of a foetal head. This had two secondary pouches, one forwards just above the valves, to the left of the pulmonary artery, the other directly towards that vessel, the coats of which were thinned and yellow, and had given way at one point just above the pulmonary sigmoids, so that there was a very small, and at present quite insignificant, recent communication between the aorta and pulmonary artery. The aneurysmal sac extended forwards to near the innominate commencement and backwards quite into the trachea, the rings of which were distinctly to be felt through the posterior walls

of the sac. In front it was well covered by the lung and adherent pericardium. The whole aorta was exceedingly diseased.

Liver 64 oz., a little granular and decidedly nutmeggy. *Spleen* 10 oz., very hard. *Kidneys* 15 oz., much indurated.

CASE 4. *Dyspnœa nine months; cardiac pain four months; palpitation just before admission; pulsating tumour to left of sternum; gradually getting worse for five months after admission; much worse three days before death. Aneurysm opening into pulmonary artery; aperture not recent.*

(Reported by Mr. E. B. GRANGER).

Alfred W—, æt. 39, was admitted under Dr. Moxon's care November 25th, 1876. He is an iron-plate and zinc worker. Has been a pretty free beer drinker; rarely took spirits. He had rheumatic fever when thirteen years old; a venereal sore at seventeen, but never any sorethroat or eruption. For the last nine months he had been low-spirited, languid, and has suffered from shortness of breath. He has for many years suffered from dyspepsia, and has often had a pain down the sternum. For three or four months he has had pain over the region of the heart. Six weeks ago he had increased weakness, dyspnœa, and pain in the left side; and his attention was first seriously drawn to these symptoms about this time by exhausting and distressing effects produced in him while playing a game of skittles. Five weeks ago he came among Dr. Taylor's out-patients and was told to give up work. Yesterday he had severe palpitation for the first time.

On admission, a well-made, dark-complexioned man; complaining of weakness, short breath, slight cough, pain over the heart. On examining this region, the præcordial dulness is found to begin in the third space. The apex beat is in the fifth space about one inch outside the nipple line; there is also a heaving impulse to the left of the sternum and marked thrill in the second and third left intercostal spaces. There is a very loud blowing systolic murmur, and a fainter diastolic one. The systolic murmur is heard all over the front of the chest, distinctly in the left axilla and less distinctly behind.

This murmur can also be heard in the carotids, and thrill is felt in the right subclavian and both carotids, but not in the left subclavian. All the arteries pulsate visibly; there is no difference between the two radials. The veins of the neck pulsate.

The lungs are apparently normal. Hepatic dulness begins at the fifth space and extends one inch below the ribs; there is some tenderness across the upper part of the abdomen. Urine is rather dark, sp. gr. 1026, clear, no albumen or sugar. His appetite is good, bowels rather constipated. Temperature normal, pulse 96.

He was treated by restricted diet, namely, twelve ounces of fluid, three ounces of meat, and eight ounces of bread daily. He had a small dose of morphia at night and ammonia and senega three times daily.

He appeared to bear the low diet well.

On December 27th it is noticed that the heaving impulse at left side of sternum is considerably less; the thrill is also less marked. He has not slept well for the last two or three nights. The pulse during the week varied from 80 to 90.

January 15th, 1877.—Has not been so well the last two days; has had slight cough. His sleep is always disturbed and unrefreshing. No increase of the impulse or murmur. Pulse 78, rather feeble.

February 5th.—Impulse and thrill are less; systolic and diastolic murmurs somewhat less loud. The former is of rougher character than it was on admission.

6th.—Was ordered twenty grains of potassium iodide thrice daily.

19th.—For the last two or three nights has had great pain in the soles of the feet, which has been scarcely relieved by poulticing and aconite liniment.

22nd.—Still suffers much from pain in the feet; morphia injection stops it for about fifteen hours, but it then returns as severely as before. Has a slight dry cough, with a little blood in the sputum. There is still a loud murmur over the whole cardiac region, and a heaving impulse in the third left intercostal space half an inch above the nipple.

During the next month he continued much the same; he was much troubled with want of sleep, and the cough and

dyspnoea became more troublesome. Morphia, chloral, and hyoscyamus were given in different combinations.

On March 1st it is noted that there was a thrill on the left side of the chest reaching from the lower border of the second rib to the fourth rib, and laterally from the middle of the sternum to one inch outside the nipple line. It was stated to be less marked on the 13th and 16th.

The last week in March he had tonsillitis, from which he recovered under astringent applications.

April 6th.—Dulness over right chest in front from second intercostal space to base; pleuritic rub from second rib to fourth space. Tactile vibration absent below third space. Posteriorly: dulness; tactile vibration absent below inferior angle of scapula; mucous râles at the base, and fine râles from apex to spine of scapula. Cardiac sounds well heard at the centre of the lung; temperature normal, pulse 120.

7th.—Right lung: dulness as before in front; pleuritic rub all over the front. Posteriorly: dulness with abundant moist râles. Left lung: impaired resonance with moist râles from the base to one inch above the angle of the scapula; above this, puerile breathing. Liver enlarged, reaching to within one inch and a half of the umbilicus, tender. Expectorationes currant-red gelatinous substance in small quantity. Pulse 120, resp. 24.

12th.—Râles on both sides of the chest back and front. Small quantities of currant-red expectoration.

16th.—Shortness of breath and sense of oppression in the chest. Continued dulness with impaired vocal fremitus at right base and also ægophony.

19th.—Troublesome cough; expectoration the same; disturbed rest. Pulse 120, resp. 26, temperature normal.

On April 23rd he died, but it is further stated in the post-mortem record that "about three days before death he was suddenly taken with shortness of breath and became very livid."

Post-mortem (by Dr. Goodhart).—The right pleura contained many ounces of fluid, and the lung was much compressed by it. There was also a layer of lymph on the surface from a small recent pleurisy. The left pleura was healthy. Right lung airless from compression. Left lung

showed a good deal of induration as in cases of heart disease. Larynx healthy. The pericardium contained 8 oz. of straw-yellow fluid, of sp. gr. 1016, and much recent lymph. At the upper part of the heart was a soft, thin-walled tumour or sac bulging into the pericardium, and above it just commencing adhesion to the sternum. This proved to be an aneurysmal sac extending upwards in the ascending aorta from the aortic valves nearly to the innominate. The whole of the ascending arch was not involved, but the sac bulged over the sound part, which thus came to open in the posterior wall of the sac. The sac occupied all the circumference of the aorta at its lower part, but the bulging took place more particularly forward and towards the pulmonary artery. The wall was remarkably thin though very diseased, and the sac contained no laminated clot whatever. On the inner side of the sac was an opening 5 mm. in diameter, communicating with the pulmonary artery behind the left posterior valve. The edge was rounded and a little thickened, the aperture slightly oval from above downwards. On opening the pulmonary artery, the aneurysmal sac was found to bulge towards it; the two posterior valves were quite useless, and adhesion had taken place in great part of their extent to the artery wall, so that they were quite unable to float out and stop the back stream of blood into the right ventricle. What remained of the valves unadherent was of quite healthy texture, without thickening either of the edge or body. The anterior valve was healthy. The aortic valves were considerably thickened at their free edges, but practically would have done fair work. They were, however, much stretched. There was some thickening of the endocardium below the valves (? from regurgitation). The right ventricle was thick, and dilated; the left thick also, but not dilated. Mitral valve good. The aorta beyond the arch was very fair indeed; the carotids, subclavians, and femorals were also good.

Stomach.—Mucous membrane intensely injected, with much thick glairy mucus and many small ulcers in the mucous surface.

Liver.—53 oz., a little nutmegged.

Spleen.—7 oz., contained a small walnut-sized calcareous cyst, full of paste (? old hydatid).

Kidneys.—12 oz., indurated heart kidneys.

Other organs healthy.

CASE 5. *Twelve months ill ; pain at chest after work ; dyspnœa ; cough. Right lung consolidated ; localised murmur not situated over valves ; increasing anasarca ; death. Aneurysm opening into pulmonary artery ; endarteritis in the latter opposite the aperture.*

(Reported by Mr. J. J. PRENDERGAST and Mr. C. H. L. MEYER, M.B.)

Charles P—, æt. 40, was admitted under Dr. Moxon, on February 6th, 1882, and was subsequently under Dr. Taylor's care.

He has worked in tanyards for the last twenty years, and has to carry heavy "butts" of hides on his head ; they weigh 80 or 90 lbs., and the work requires great exertion.

He has never had rheumatic fever ; has never been laid up before now ; he had gonorrhœa fifteen years ago, but denies syphilis. He is married, and has five children. He has taken a good deal of beer daily, and has often been drunk. For several years he has had a slight cough, but without expectoration, and has never spat blood. For the last twelve months he has noticed that after his day's work he has had pain across the lower part of the front of the chest, and shortness of breath ; but the pain never came on during his work. It used to last a quarter of an hour, and was relieved by bending the body and by rest.

On January 24th, a very foggy day, he went to work at 5.30 a.m. in his usual health. He worked until 8.30, when he had to leave off on account of constant coughing, distressing shortness of breath, and sharp shooting pains through the left chest. He kept at home, and on the 25th and 26th was sick at the sight of food. He came to the hospital on the 27th, but did not come in, though recommended for admission.

On the 29th he again came as an outpatient, and had some medicine which relieved the cough somewhat, making the expectoration more free.

On February 5th he was again recommended for admission on account of dyspnœa, pain, and cough, and he came in the following day.

Present condition (February 7th).—A tall, strong, well-nourished, muscular man, with an anxious expression, rapid

breathing, frequent coughing, and pain in the left side, over the region of the heart. The heart's impulse is feeble, and diffused. A systolic murmur is heard at the apex, and a diastolic at the base; and a pericardial rub is also present. Pulse regular, compressible, splashing, 116. The left chest is resonant on percussion with loud vesicular murmur; the right is dull in front and behind, with crepitation over the upper lobe of the right lung from clavicle to nipple, and moist sounds at the base of the same lung behind. Vocal fremitus is normal on both sides. He spits much tenacious, viscid, rust-coloured phlegm, which adheres to the vessel when inverted. Brassy cough. Resp. 40.

Tongue large, flabby, dull red, not furred. Thirst; anorexia. Bowels regular, once daily.

Liver reaching to umbilicus; spleen normal. Urine high-coloured, sp. gr. 1030, free from albumen and sugar. He was ordered ammonia mixture; middle diet without beer.

February 9th.—Improved. Sputum still rusty. Respirations about 30; lies on right side. Dr. Moxon found at the base of the right lung much dulness. Deficient entry of air. Tubular breathing, and bronchophony.

11th.—Right chest in front slightly dull with small crepitation just above the nipple; behind much dulness with mucous râles.

During the rest of this month he continued to have dulness at the right base behind with moist râles, and spat varying quantities of rusty-coloured sputum. The temperature was never taken above 99°. On the 16th he was ordered five grains of iodide of potassium, with acetate of potash, squill, and nitrous ether, three times a day, and his back was painted with tincture of iodine. On 25th Dr. Taylor noted that there was no murmur with the second sound, but that it was somewhat roughened, and that the pulse had the characters of aortic regurgitation to a certain extent.

March 2nd.—In twelve hours, three ounces of sputum have been expectorated, for the most part rusty and watery, partly tenacious, and green.

R. Pot. Iodid., gr. v;

Ex. Mist. Cinchoninæ, ℥j. T. d. s.

4th.—Was up for two hours, feeling none the worse for it.

6th.—He feels better; there is very little expectoration.

Dulness and large crepitation behind at both bases, especially the right. Vocal fremitus good. Both apices normal. Iodine application continued.

7th.—Bowels open three times. When he gets up in the evening he walks about the ward with ease, not being nearly so short of breath as he was before admission. But he feels pain in the region of the heart, and suffers from panting whenever he stoops.

9th.—The condition of the right base is the same, but the sputum is less in quantity. He went down into the grounds, and was very short of breath; on returning he was unable without assistance to get up all the steps, suffering much from dyspnœa and pain over the heart. After this he continued short of breath, and his legs being found œdematous on the 11th he was again confined to his bed. Pulse 100, resp. 26, shallow, and sometimes sighing.

13th.—His face is puffed this morning, and the œdema extends up his legs to the middle of the thighs.

14th.—Dulness at right base, with crepitation, tubular breathing, but no bronchophony. Ordered jalap purge and diuretic mixture with digitalis.

During the next fortnight the physical signs at the right base continued much the same, except that in a diminution of the bronchial breathing and vocal resonance, they more resembled those of pleural effusion.

The œdema of the legs increased, and extended to the hands and arms, penis and scrotum; and there were signs of fluid in the abdomen. An examination of the heart on April 1st gave the following results:

The impulse was felt over a wide area corresponding to the cartilages of the left third, fourth, fifth, and sixth ribs, and extending outwards to just beyond the nipple. A murmur was heard over the sternum, and widely on both sides of it, but its greatest intensity was to the left of the sternum, near the third and fourth cartilages, and corresponded to neither aortic nor pulmonary valves, nor to the tricuspid orifices.

The second cardiac sound was heard most distinctly in the third space about an inch from the sternum; there was slight dulness at the upper part of the sternum as low as the third cartilages. His face flushes very much on the slightest exer-

tion. He has no pain, but retches often. Pulse 110, resp. 26. Temperature last night 97.8° . He has been taking for the last ten days five minims of tincture of digitalis in copaiba resin mixture, with occasional doses of compound jalap powder.

April 6th.—A grain each of squill, digitalis, and blue pill given three times a day.

His anasarca increased, he became more livid, he vomited frequently, and the cough became troublesome, with expectoration of blood-tinged mucus. The legs were punctured on April 10th. There was a good deal of albumen in the urine on the next day, when the following note of his condition was also made.

At the right base dulness, absence of vocal fremitus, and deficient breath-sounds with very few crepitations. Cooing expiration at both apices, slightly increased vocal resonance at the right apex with prolonged expiration. Very marked pulsation in suprasternal notch, with distinct bulging on coughing; well-marked dulness over manubrium, and upper sixth of gladiolus, in which latter situation the sternum appears to bulge forward a little.

An exploratory puncture was made at the right base, and fluid was found. The patient is heavy and sleepy, but looks less anxious. He had some diarrhoea in the night.

12th.—He is very much worse; the slightest movement causes great dyspnoea. The sputum is abundant, frothy, blood-coloured. Frequent troublesome cough. Great and increasing dyspnoea; much oedema; yellow tinge of conjunctivæ. Pulse 144, very splashing. Increasing crepitation in the right lung with pleuritic sounds at base in front. Brandy and ether internally, subcutaneous injection of ether, and inhalation of nitrite of amyl failed to rally him, and he died at 9.40 p.m. His temperature, which had never reached 99° , rose on April 7th to 101.2° , and remained between 99° and 101.5° until his death. The urine contained albumen varying from a trace on April 2nd when it was first observed to one eighth on the day of death.

Post-mortem (by Dr. Goodhart).—Jaundice; dropsy of scrotum and legs.

Right lung partly hepatised, the colour more brown than usual, and some solidification of left lung also.

Larynx a little œdematous.

Pericardium healthy.

Heart not weighed, but must have been 15 or 16 oz. at least. Both ventricles dilated and hypertrophied.

On slitting up the pulmonary artery a bulging swelling was noticed on the right side of it, occupying about half its calibre, immediately above the pulmonary valves. In the posterior half of this was an elongated slit which had rounded edges, and was evidently of old date. On the face of the pulmonary artery, which corresponded with this swelling, was a patch composed of slightly raised ridges arranged in an annular form, and within these a separate *plaque* of similar nature, only that it had a more continuous surface. At first this was regarded as due to the rubbing of the surface of the aneurysm against the opposite wall of the vessel; it was, however, probably due not to this, but to the propulsion of a jet of blood from the sac into the artery. If so this is a case which proves that the blood made its way from left to right when the separation between the two circulations was rendered incomplete. And although it is nearly certain that this must always be the case, yet there are not many cases which give the required demonstration. The sac described was an aneurysm of the aorta, which came off from the latter just above the left anterior valve, and opened from the aorta by an orifice half an inch in diameter. It was about one inch in diameter itself, and contained some recent blood-clot.

The aorta was extremely diseased throughout, indeed nowhere healthy, and the disease consisted in the conversion of the coat into thin grey patches with some yellow ones. There were many aneurysmal pouches smaller than the one described. One was half an inch in diameter, and pouched into the pericardium, and was at one spot so thin that it must have ruptured within a day or two. The finger showed through it distinctly, and it was almost like tissue-paper.

There were three or four others in various parts of the thoracic aorta, and there was another of considerable size at the commencement of the superior mesenteric artery. The whole aorta here was involved, and the two renal arteries were somewhat implicated.

The aortic valves were considerably thickened, and let down

from their normal attachments for about one third of an inch ; but they appeared to hold water now very well, nor were there the usual striations of the endocardium below them, indicating regurgitation.

Liver.—55 oz., very nutmegged, but not granular nor indurated.

Gall-bladder.—Full. *Spleen.*—3 oz., very firm. *Kidneys.*—12 oz., healthy. *Testes.*—Healthy.

CASE 6. *Four months ill ; giddiness and dyspnœa on exertion ; pain in chest ; large heart ; loud roaring bruit ; much lividity ; anasarca ; death. Aneurysm of aorta opening into pulmonary artery.*

(Reported by Mr. MUIRHEAD.)

Thomas F—, æt. 45, was admitted under Dr. Goodhart on August 9th, 1882, for pains in the chest, swelling of the legs and shortness of breath. He had been a soldier in the Royal Artillery, and was now a labouring man. With the exception of gonorrhœa when a young man, he has been quite free from illness, until five months ago ; and he denies syphilis, and drinking. In March of this year he had some very severe work, and about three weeks after it he noticed that on the slightest exertion he became giddy, and had shortness of breath and severe pain in the chest and left side. He was sometimes so bad that he could not go on with his work, and could not walk home. The trouble has continued ever since.

Present condition.—He is well nourished, 5 ft. 6 in. high, and weighs 10½ stones. The skin is yellowish, moist ; the face flushed ; the legs are swollen. The heart's impulse is plainly visible and diffused ; the præcordial dulness increased, and extending a little outwards towards the nipple. At the base there is a loud blowing systolic murmur, audible down the sternum below the middle, but not traceable into the neck. At the apex there is a sharp systolic murmur, not always audible ; this is also heard behind at the angle of the scapula. Pulsation of the vessels in the neck is very perceptible, but no bruit is heard in the carotids. The radial pulse is full, splash-

right of the face
 yellowish
 all the opening
 in; and this was
 right side was
 left side above
 The first inch or
 more defined by
 some anastomosis,
 all connection
 valves were par-
 te, and through
 recently formed,
 elongated trans-
 as an inch above
 the aorta, ventral
 is formed by the
 evidence in this
 is into the pri-
 mary of the
 o the left of the
 vessel, have been
 in. On opening
 in diameter was
 very artery. I
 ed no sign of any
 y disease, being
 e were two other
 e surfaces. The

e mitral disease.

ing, 104. The chest is equal on both sides, and there is good resonance with normal vesicular murmur, vocal and tactile fremitus on both sides. The respiration is full when he is at rest, but hurried when he moves. He has a troublesome cough, and scanty muco-purulent sputa. His voice is clear. Resp. 20. Urine acid, sp. gr. 1025. Albumen $\frac{1}{2}$. The abdomen is normal, the liver dulness not increased; bowels regular; no vomiting or nausea. Temperature normal. Ordered senega and ammonia every four hours.

On August 12th he was very ill; skin moist; face very flushed; increased oedema of legs. Urine, sp. gr. 1028, albumen $\frac{1}{2}$. At 3.15 p.m. he became suddenly worse, with lividity and dyspnoea. The pupils dilated, contracted, and again dilated; the dyspnoea increased, and he died.

The following is Dr. Goodhart's account of the post-mortem, with his notes of the condition of the heart and vessels during life:

"Cardiac dulness diffused, slight pulsation at the base of the heart, seen rather than felt. Much lividity, but the veins of the neck did not pulsate nor were they distended. At the base of the heart to the left of the sternum, over the region indicated, was a peculiarly roaring musical bruit, so prolonged as to occupy the entire systole, and run one systole into another. The second sound was barely audible. There was no diastolic bruit, and no thrill. From the character of the bruit I considered that an aneurysm existed above the valve, and from his lividity and general anasarca, I thought it probable that a communication existed between the pulmonary artery and the aorta.

"Pleuræ, some old adhesions on both sides; both lungs oedematous.

"The pericardium contained 2 or 3 oz. of fluid; when the sac was opened, an ovoid bulging of the aorta came into view, to the right of, and partially overlapping, the first part of the pulmonary artery. When the pulmonary artery was opened it was found that the right wall of the artery was bulging inwards; the cusp of the pulmonary artery on this aspect was, though still distinguishable with care, firmly adherent to the bulging arterial wall behind; one of the other cusps was partially adherent, and on what may be called the hinder wall

—on the deep aspect of the bulge—there was a small circular opening, $2\frac{1}{2}$ mm. in diameter, from the aneurysm which formed the projection into the pulmonary artery. This opening was opposite the posterior cusp, against which it had obviously allowed blood to jet, for the surface of the cusp was both roughened and ulcerated. The right ventricle was very much hypertrophied, its walls reaching a thickness of about 8 millimetres.

“The aorta just above the valves was not so very bad, but a patch had given way, on the inner aspect of the arch which had bulged into an aneurysm with an orifice $\frac{2}{3}$ inch in diameter. The aneurysm was about $1\frac{1}{2}$ inches in diameter, and its walls were lined with an adherent laminated clot of no long standing, to judge from the want of decolorisation.

“The thoracic and abdominal portions of the aorta were much diseased, their coats being blotched all over with yellow and grey atheromatous patches. The cavities of the heart, including the left ventricles, all full of blood. The aortic valves were a little thick, but practically healthy.

“*Liver*.—49 oz., nutmegged. • *Spleen* hard, not lardaceous. *Kidneys* congested; one had a fibroid in one of the pyramids.”

CASE 7. “*Heart disease*” *six years (?) ; recently epigastric pain ; anasarca ; systolic murmur ; dyspnœa ; collapse ; death ; aneurysm communicating with pulmonary artery ; one recent and one old opening ; a third opening doubtful.*

(Reported by Mr. C. FRYER.)

Joseph E—, æt. 48, was admitted under Dr. Pavy, October 26th, 1888. He was by occupation a seaman. He had measles as a child ; twenty years ago he had a hard chancre and suffered from pains in the joints afterwards, but had no sorethroat and no rash. Sixteen years ago he was in a French hospital for an illness which he thinks was enteric fever. He has had no rheumatic fever or bronchitis. About six years ago he had a violent cold, and was told by a doctor whom he consulted that his heart was affected. He was very feverish and had rigors, and a cough, which was worse at night and

prevented him from sleeping. He did not expectorate much, but had a feeling of great tightness across the chest. He passed an ordinary amount of urine of dark colour; his bowels were regular. A few weeks ago he noticed pain in the epigastrium, which was worse when he took a deep breath, and lately worse after food. Recently he has felt weaker, has taken to his bed, and his legs have swelled. There has been no swelling of the scrotum or the eyelids.

On admission he is pale, with a cold, moist, very pale skin; he breathes with difficulty, is unable to lie down; has slight œdema of the legs. Pulse 135, temp. 99°.

The heart's dulness commences at the fourth rib and extends to the area of the liver dulness. The apex beats in the fifth space one inch below and half an inch external to the nipple. A systolic bruit is heard at the apex. There is marked pulsation in the triangles of the neck and in the suprasternal notch. The tongue is slightly furred and dry, and he complains much of thirst. The liver dulness extends from the fifth rib to the margin of the thorax. The urine is scanty, high coloured, of sp. gr. 1028, containing albumen and a slight trace of sugar, but no blood.

On the night of October 28th the patient had much dyspnœa and became cold. The following morning he was unable to lie down in bed, his hands were cold and clammy. He had much dyspnœa, and the heart's action was very quiet. During the morning he vomited at intervals. At 3 p.m. 7½ oz. of urine were drawn off by the catheter, it was of dark colour, sp. gr. 1012, very acid, and contained albumen. Later on he was much collapsed, with cold extremities, and forehead covered with perspiration; the pulse, however, was regular and compressible. He became drowsy, and with gradually increasing weakness died at 10 a.m. on the 30th.

Post-mortem (by Dr. Goodhart).—*Brain*.—55 oz., healthy.

Lungs.—Both bases much consolidated, partly by fluid in the pleura, which, however, was not much, partly by hypostatic pneumonia, and partly by œdema.

Larynx healthy.

Heart.—The pericardium contained 2 oz. or so of flaky lymph. The membrane was minutely injected and dull, but no distinct lymph could be seen upon it. The anterior sur-

face of the heart showed immediately to the right of the root of the pulmonary artery a small bulging with yellow spotting about it, which looked very like an aneurysm, all but opening into the pericardium from the root of the aorta; and this was subsequently found to be the case. The right side was rather large, and flabby and dilated. The left side about normal, relaxed, but probably not dilated. The first inch or inch and a half of the pulmonary artery was much defaced by inflammatory change in its coats due to an aortic aneurysm, which had thinned the coat and destroyed all distinction between it and the aorta. The pulmonary valves were partially adherent from the growth of the sac, and through them was an opening which had apparently recently formed, and was fringed with vegetations. It was elongated transversely, about 6 mm. by 2 mm. in size. About an inch above the valves was another larger opening into the aorta, vertical in direction, about 12 mm. by 2 mm., with rounded edges and evidently of old date. There was no evidence in this case of the passage of blood from the aorta into the pulmonary artery by the presence of any thickening of the opposite arterial wall. Below and a little to the left of the last was a third opening, which may, however, have been caused by a tear where the coats were very thin. On opening the aorta, an aneurysmal sac about an inch in diameter was found just about the orifice of the left coronary artery. Its walls were very thin and grey, and it contained no clot of any kind. The aorta for the first inch was very diseased, being converted into a thin grey material, and there were two other aneurysms commencing in depressions of the surfaces. The valves were as yet quite healthy.

Intestines healthy.

Liver "nutmeg."

Spleen and *kidneys* indurated as in chronic mitral disease.

Suprarenal capsules healthy.

ON

HERNIA OF THE APPENDIX VERMIFORMIS.

By N. DAVIES-COLLEY, M.C.

PHYSIOLOGISTS do not appear to have decided whether the vermiform appendix should be looked on merely as a vestigial structure, or as having some especial function, such as the lubrication by its glandular secretion of the adjacent cæcum, in which fæcal matter is apt to accumulate. There is no doubt, however, that it is often the cause of serious danger from the proneness of foreign bodies or fæcal concretions to lodge within its canal, and numerous cases are on record in which such an accident has given rise to ulceration and perforation, with extensive suppuration or even fatal peritonitis. But I am not aware that any of the writers of our surgical text-books have called attention to the possibility of severe symptoms arising from the incarceration or strangulation of this process within a hernial sac, although several instances of this condition are to be found in the periodical literature of this and other countries. It is by no means a rare occurrence to find in a large inguinal hernia of the right side that the cæcum has descended, accompanied by its appendix ; but I allude now to cases in which the appendix alone has formed the contents of an inguinal or femoral hernial sac.

There are also a few recorded cases in which the end of the appendix has become adherent to the adjacent surface of the bowel or mesentery, and so formed a loop through which portions of bowel have passed and been strangulated, either in the interior of the abdominal cavity or within a hernial sac.¹ These, however, do not come within the scope of this paper.

The first of my cases is one of acute strangulation of the appendix. It is a good example of the advantage of operating early even though some of the symptoms of strangulation are absent. If, on account of the action of the bowels and the diminution of the vomiting I had decided to wait, I think that the appendix would have become inflamed and adherent, so that later on I should have had to deal with a more serious condition, such as was observed in some of the cases to be subsequently described.

CASE 1. Right femoral hernia containing appendix vermiformis ; strangulation ; herniotomy ; recovery.

Millicent M—, æt. 38, was admitted into Guy's Hospital, under my care, on October 2nd, 1883. She was a married woman and had had eight children, the last nearly two and a half years ago.

Twelve months ago she was lifting a heavy basket when she felt a sudden pain in the right groin followed by an attack of vomiting. These symptoms soon afterwards subsided but from time to time they recurred. On the second occasion she felt a small lump in her groin. She was always able to return it, and the pain never lasted more than an hour. Sometimes it would be two or three months before it came down again. She never wore a truss.

¹ Dieffenbach ('Operative Surgery,' vol. ii, page 571) describes a case in which upon opening the sac of a large inguinal hernia, he found the cæcum strangulated by an adherent appendix. The patient recovered. He also quotes another case from Scarpa, in which a loop of ileum was similarly strangulated within the abdominal cavity. In the 'Pathological Transactions' (vol. iii, page 101) there is an account of a patient who had repeated attacks of internal strangulation, and in whom after death it was found that the constriction was due to a ring of which one part was formed by the appendix with its extremity adherent to the ileum. In a previous volume of these reports (Series iii, vol. xiv, page 358), Dr. Hilton Fagge calls attention in his paper upon intestinal obstruction to a similar case, and he refers to three specimens in our museum illustrating the same condition.

On Sunday evening, September 30th, when in chapel, about 6 p.m., she had a sudden attack of the pain and felt the lump in her groin, but when she got home she was unable to return the hernia as before. Vomiting began about 9 o'clock, and went on during the following day, so that she could retain nothing upon her stomach. The matter vomited was not stercoraceous. Taxis was tried by a medical man on the evening of October 1st, with considerable pressure, and ice was then applied. The next morning, October 2nd, he again used taxis without success, and he advised her to come to the hospital. Her bowels were moved once on the 30th, twice on the 1st, and again slightly on the 2nd.

On admission.—She had a somewhat apathetic appearance. Her pulse was feeble, and her skin cold. There was a swelling over the right femoral ring, of a globular shape, one to one and a quarter inches in each direction. It was rather movable, and an attached neck could be made out somewhat indistinctly. There was no impulse upon coughing. It was thought to be possibly an enlarged gland but more probably a strangulated hernia, consisting either of omentum or of a part of the calibre of the bowel.

Ether having been administered I made an incision through the integuments, and after a short dissection a congested sac was exposed and opened. In it the appendix vermiformis lay coiled up. It was about three inches long, of normal thickness, and but little altered in appearance, except that one inch from the tip there was a blood-clot under its peritoneal covering measuring three eighths of an inch in its longest diameter. Having notched the internal border of the femoral ring I readily returned the protrusion. I then brought together the sides of the sac with catgut sutures so as to obliterate its cavity, and I put wire sutures in the margins of the skin incision. The usual antiseptic precautions of spray, gauze, &c., were adopted. There was no fluid nor omentum in the sac, the walls of which were reddened and rather thick.

The patient made a good recovery. On October 3rd and 4th her temperature rose to a little over 102°; it then fell to normal. Some suppuration followed the operation but the wound was healed by the 21st. She was allowed to get up on the 25th, and she went out in good health wearing

a truss upon the 3rd of November, thirty-two days after the operation.

My second case is an example of the condition which is apt to follow the prolonged incarceration of the appendix in a hernial sac. In such a case it is difficult to say what is the best course to pursue. Are we to leave the inflamed process in its place, after dividing the constriction at the neck of the sac, or should we attempt to return it, or, thirdly, ought we to remove it as I did in the following case?

CASE 2. *Strangulated inguinal hernia ; partial reduction ; operation ; removal of appendix cæci and omentum ; peritonitis ; death.*

Samuel A—, æt. 58, fish curer, was admitted into Astley Cooper Ward, Guy's Hospital, on January 21st, 1880. He has had a right inguinal hernia thirty years. Latterly his truss has become inefficient, and part of the rupture has always remained down. On the 17th it became larger, and on the 20th taxis was first applied. There has been no passage of motions or flatus since the 18th. He has not vomited. I first saw him at 5 p.m. on the day of his admission. He had a hard red tender swelling of a cylindrical shape in the right inguino-scrotal region about five inches long and two inches in diameter. There was no impulse on coughing. After taxis had been applied gently but firmly for five minutes, a gurgle was felt and the swelling became smaller. For a short time longer gentle taxis was used, but no further reduction of the swelling was effected. Ice was then applied. In the evening he passed some flatus.

22nd.—Still low and feverish, and swelling nearly if not quite as large as when he came in. Had passed some flatus. In the afternoon ether was administered, and I explored the contents of the still swollen sac. There was no fluid in its interior, but a quantity of omentum which was swollen and matted together, and with some difficulty separable from the sac. Under cover of this and rather to its inner side, was a cylindrical mass with a rounded extremity, the whole about three inches long and one inch thick. Upon its surface was a

longitudinal depression or constriction, which made it resemble a loop of very much contracted bowel, of which the descending and ascending parallel portions had become closely blended by their adjacent surfaces. It was of a reddish-purple colour, and there were spots of yellowish lymph beneath its shiny covering. Nothing else was found in the sac. Considering that this mass, which I looked upon as an inflamed appendix cæci, was causing great constitutional irritation, and that if left in the sac it would prevent the proper application of a truss, and be a constant source of pain to the patient, I decided to remove it. With a strong silk ligature I secured its base, and then cut off from two to two and a half inches. The omentum was similarly tied and removed. The edges of the wound were then brought together with silver sutures. There was a hydrocele on the same side about as large as a goose's egg, which I left alone. The operation was performed antiseptically. There was a little vomiting the following day.

24th.—Temp. 97.4° .

25th.—Pulse 88, temp. 98.5° . Tongue dry and brown. Third dressing. Half an ounce of bloody serum to-day without smell; good apposition of wound, no redness, no increase of swelling. There has been no return of the vomiting. Has taken *Pil. Opii gr. j ter die*, till to-day. To-day pills ordered to be taken only at night. Brandy \mathfrak{z} ij.

26th.—Pills stopped. In the morning pulse 92, temp. 99.1° ; in the evening pulse 92, temp. 100.5° .

27th.—Some smell in dressings. In the morning pulse 92, temp. 99.2° ; in the evening pulse 108, temp. 100° . Enema, after which bowels acted well. Abdomen full and prominent.

28th.—Mist. *Sp. Vini Gallici* \mathfrak{z} j *secundis horis*. In the morning pulse 106, temp. 100° , resp. 40.

29th.—In the morning pulse 108, temp. 99.8° , resp. 44. Patient wandering.

He died on the 30th, nine days after his admission, and eight after the operation.

The post-mortem examination was made by the late Dr. Hilton Fagge.

The lungs were very bulky, and full of points of broncho-pneumonia, not unlike tubercles beginning to caseate, but softer

and less defined. The heart was hypertrophied, and the aortic valves were much thickened and adherent.

There was acute peritonitis, the coils of the intestine at the upper part of the abdomen being adherent by a small quantity of recent lymph. In the pelvis the inflammation was of older date, in connection with a coil of ileum which had evidently been strangulated in the hernial sac, as it was deeply congested between two definite borders. The cæcum was greatly distended and prominent, and its pouch was in contact with the internal abdominal ring, so that while it was *in situ* the condition of the parts could not be made out. But after removal, and when the cæcum had been emptied of the gas and thin faecal matter which it contained, it appeared that the only part which passed down into the inguinal canal was the appendix, the funnel-shaped orifice of which could be seen just above the ring. The peritoneal pouch which formed the sac was in front of the appendix. Both were abruptly truncated in the wound made in the operation, and each had a ligature round it. But below these truncated ends there was a remarkable fleshy mass which ran down to the testicle. This when laid open proved to consist of a soft whitish-yellow curdy material, which Dr. Fagge thought could have been nothing but an inflamed spermatic cord infiltrated with fibrinous exudation. The tunica vaginalis contained a little fluid. The testis itself was healthy. The kidneys also were healthy.

In this case it would appear that the fatal result was not due to the removal of the inflamed appendix, but to the injury which had been received by the strangulated small intestine, and the peritonitis which it had set up after its return to the abdominal cavity. It is probable that the incarceration of the omentum and appendix had kept a way open by which the small intestine had been able to escape from the abdomen, and had interfered with the proper application of a truss to prevent this escape. The ligature and removal of the appendix do not seem to have been followed by any severe symptoms, and there is good reason to suppose that, but for the injury already sustained by the small intestine, the patient would have made a good recovery.

My third case, which Mr. Durham has kindly permitted me

to publish, indicates another result which may follow acute strangulation of the appendix. In this instance an abscess rapidly formed, and after exit had been given to the matter recovery took place with a fæcal fistula, which was still discharging slightly when the child was last heard of.

CASE 3. *Strangulation of the appendix vermiformis in an infant ; recovery with a fæcal fistula.*

(From Report by Mr. H. P. BERRY.)

James H—, æt. 5 months, was admitted into Guy's Hospital under Mr. Durham, on March 5th, 1881. He had had an ordinary reducible inguinal hernia on the right side since birth. This had gradually increased in size. Three days before his admission his bowels had not acted and he had suffered from vomiting, which had latterly become almost incessant. The day before his admission it was found that the hernia could not be reduced, and in the evening the swelling became red.

On admission he was found to have an irreducible and very tense swelling in the right groin. The adjacent side of the scrotum was red, œdematous, and tender, and the right testis was concealed, if present, by the œdema. There was no swelling of the abdomen. He was passing a good deal of flatus per anum.

Chloroform was administered, and Mr. Durham then proceeded to operate. The sac was laid open. No constriction was found, but the hernial sac was suppurating. About twenty drops of pus escaped, and nothing else was seen in the sac except a portion of the appendix vermiformis which was sloughing and perforated. Mr. Durham was able to pass his finger with ease into the abdominal cavity.

March 6th.—The child has slept fairly well, and seems to be quite cheerful. Has vomited once this morning, and has had a good action of the bowels. He takes the breast well. Morning temp. 97·5°.

7th.—No return of sickness. Bowels have been twice moved. Rather more fretful. Has only slept at intervals. Morning temp. 97·9°. Pulse 124. Poultices applied.

8th.—Temperature 102°.

10th.—Rather fretful. Morning temp. 99·8°, pulse 172. Evening temp. 99·4; pulse 152.

12th.—Temperature 100·4°.

14th.—Very bright and cheerful.

18th.—Seems well. Temperature normal for the last day or two.

19th.—A trace of fæces in the wound. Patient was costive, and had been straining to pass a motion. Temperature normal.

20th.—Fæces have not come through the wound again. The hardness of the swelling is subsiding.

24th.—Temperature still normal. The child was allowed to be taken out of the hospital. There was still a small discharging sinus, which was dressed with carbolic oil.

For a long time the child was kept under observation. The sinus remained open for more than a year, and there was an occasional discharge of fæcal matter from it.

I am indebted to the kindness of Mr. Clement Lucas for permission to publish the report of the following case which has recently occurred within our walls.

CASE 4. *Strangulated right femoral hernia containing vermiform appendix; operation; ligature and removal of gangrenous appendix.*

(From Notes by the Surgical Registrar, Mr. POLAND, and the Ward Clerk, Mr. J. A. BENSON.)

Lucy C—, æt. 47, was admitted into Lydia Ward under Mr. Clement Lucas, on May 14th, 1884.

She is married and has had fourteen children all of whom are healthy. Latterly she has suffered from a sensation of weakness over the abdomen and chest, and she has also had bronchitis for three years.

On December 24th, 1883, after a hard day's work, she noticed a swelling in the right groin which came down suddenly and caused her great pain. A medical man was sent for who reduced the swelling, and there was no further trouble until the 9th of May, five days ago, when the hernia again came down about the size of a nut while she was walking and coughing.

On the 11th the bowels were opened, and taxis was applied twice.

On the 13th taxis was again applied. Vomiting began fifteen hours before her admission on May 14th.

On admission the patient was pale and haggard, with a yellowish skin, and an anxious expression. The pupils were contracted from the opium which she had taken. The lips were dry, the tongue moist but somewhat furred. Her breath had a faecal odour. In the right groin below Poupart's ligament was an ovoid swelling two inches in circumference. No impulse on coughing. The abdomen was tender. She had had no vomiting for four hours before admission.

Mr. Lucas was called at 7.40 p.m., and chloroform having been administered he proceeded to operate. A vertical incision two inches in length having been carried down to what was believed to be the sac, this was opened and about two ounces of serous fluid escaped. A tense knuckle was then exposed, which though covered with serous membrane had not the appearance of strangulated bowel, and some adhesions were found at the upper part attaching it to the parietal layer which had been opened. The presenting knuckle was carefully scratched through with the point of the knife till another spurt of serous fluid escaped; and this time some foetor was noticed, though the serum was not discoloured so far as could be judged under the steam spray. In the sac lay another pale grey coil, again unlike strangulated bowel. The grey layer was caught with forceps, torn, and raised, when a deeply congested, dark purple coil, at one point mottled green, was exposed. The grey layer was apparently a thick coat of organising lymph which completely obscured the bowel, or possibly the sodden and separated peritoneal coat of the gangrenous bowel beneath. The bowel exposed was about the diameter of one's fourth finger and somewhat constricted at the sides. A needle was passed from the skin through the coil with the object of making an artificial anus, when the bowel suddenly and completely collapsed with the escape of foetid air but no faeces. The shrunken loop was now recognised as appendix caeci, and on pulling it out the free end was reached. To make sure a probe was passed into its interior and it made its way without obstruction, a distance of four or five inches. A little more faecal gas then escaped but

no fæces. The appendix was ligatured at its base with catgut and cut away. The wound was left open and antiseptic dressings were applied. The patient at the conclusion was much collapsed, and it was some time before she rallied from the chloroform. She was ordered a grain of opium every four hours.

15th.—No distension of the abdomen; only slight tenderness in region of the wound. First dressing under spray. No fæcal matter about wound. Temperature $100\cdot6^{\circ}$ in the evening.

16th.—Slight abdominal tenderness. Respiration quiet. No vomiting. Cough troublesome. Temperature in the morning $99\cdot6^{\circ}$, in the evening 100° .

19th.—Slight fæculent smell about the wound.

21st.—Bowels acted a little. Dressed daily.

24th.—Opium stopped.

31st.—Bowels act regularly.

June 6th.—Right femoral truss applied.

17th.—Wound nearly healed.

July 12th.—Discharged quite well.

In addition to these four cases, I will give brief *résumés* of a few others which I have found on record.

Mr. Pick¹ has reported an interesting case, very similar in some respects to Case 2. A painter, æt. 54, was admitted into St. George's Hospital on February 21st, 1880, with a right inguino-scrotal rupture. He had had it twelve years, but had always been able to return it, and had never worn a truss. Five days before admission it came down and could not be returned. There was a painful swelling, constipation, and vomiting, which had latterly been offensive. Œdema and redness extended nearly to the umbilicus. There was no impulse on coughing. Taxis had been unsuccessfully applied. Upon operating pus was found in the sac, and an inflamed vermiform appendix. There was a tight stricture at the neck of the sac which was divided. The inflamed appendix was cleaned and pushed back; the suppurating sac was dissected out and removed. He died five hours later of local peritonitis. Mr. Pick thought that the case was originally a hernia of intestine and omentum,

¹ 'Lancet,' vol. i, 1880, page 801.

but that some inflammation had occurred about the neck of the sac and strangulated the appendix, which had happened to get into the sac.

A similar but more successful case is recorded by Mr. Josiah Court.¹ A married woman, æt. 50, had suffered from strangulated right inguinal hernia for one day. There was frequent vomiting, with constipation, and the abdomen was hard, tender, and tympanitic. After an unsuccessful attempt to reduce the swelling by taxis, cold was applied, and opiates were given. The more pressing symptoms subsided, but the swelling remained, and the bowels would not act. As the pain grew worse, chloroform was administered and the sac was opened. He found what he at first thought to be a gland, but on careful scrutiny it turned out to be the vermiform appendix firmly adherent at the neck of the sac. Having separated some adhesions and divided two bands, he was able to return the protrusion, and a complete recovery ensued.

Dieffenbach² mentions the case of a gentleman, æt. 63, who was suddenly seized with sickness, eructations, and a dragging sensation in the lower part of the abdomen. He felt generally ill, but the passage of motions was regular. On account of his leanness it was easy to ascertain that all the usual sites of rupture were free, but upon firm pressure in the region of the right femoral ring, the patient felt a slight sensation of dull pain extending upwards into the hypogastrium. After leeches, poultices, and other remedies had been tried in vain, Dieffenbach made an incision, and found in front of the right femoral ring a hard swelling, the size of a small bean, which could easily be recognised as peritoneum. Adherent to the internal surface of this membrane was a blackish-grey body, folded and grown together, which after it had been released and unfolded proved to be the end of the vermiform appendix, which had become fixed in a very narrow femoral ring. It was easily returned after a small incision had been made into Gimbernat's ligament. There was, however, no improvement in the condition of the patient. Without any relief of his earlier symptoms, he sank two days later from exhaustion.

There is an interesting account in the Pathological Society's

¹ 'Lancet,' vol. ii, 1870, page 401.

² 'Op. Surgery,' vol. ii, page 600.

'Transactions'¹ by the late Mr. De Morgan, of a man, æt. 66, who was admitted into the Middlesex Hospital with a reducible inguino-scrotal hernia, which he had had for forty years. For two weeks he had felt ill and remained in bed. Two days before admission he had tried to reduce the swelling without success. He suffered great pain in it, but there was neither constipation nor vomiting. There was a large tumour about the size of an orange in his right groin of an oval shape, tense, and unyielding to the touch. There was no true impulse on coughing. The skin was congested and the tumour very tender and painful. There was some swelling but no tenderness of the abdomen. An aperient was given, which brought away some natural motion, and the patient felt relieved. The next day he was worse, and the abdomen more swollen.

An operation was now determined on, the impression of the surgeon being that the hernia was omental, and the omentum becoming strangulated. Upon cutting through a capsule more than half an inch thick, lined with unorganised fibrin, a cavity was exposed which contained about three drachms of thick greenish pus, free from all offensive odour. In the wall of this cavity was found upon its outer side a small opening which led up to a second and smaller cavity. This second cavity or track was laid open for some distance, but a probe could be "passed on indefinitely" into it. No intestine or omentum was observed, and although during the operation flatus and soft fæculent matter escaped from the anus, none was discharged from the wound. The edges of the incision were brought together with sutures, but the patient sank rapidly and died about two hours and a half after the operation.

At the post-mortem examination it was ascertained that the smaller cavity was that of the appendix vermiformis, which was two and a half inches long, and wide enough to contain a large goose-quill. At its further end was an ulcerated opening, but no communication could be detected with the large cavity, which was the remains of a hernial sac of which the neck had been obliterated. Some fish bones, however, were found upon a careful examination of the larger cavity. On the posterior wall of the cavity the vessels of the spermatic cord were seen enlarged and dilated, and incorporated with it. Mr.

¹ Vol. xxv, p. 107.

De Morgan thought that the appendix had been in its position for many years, and that there had originally been also a hernia of the cæcum, while the thickening of the wall of the larger cavity was attributed by him to the fact that it was formed by altered omentum. He considered that the death of the patient was due to congestion of the lungs, which were loaded with blood and serum. It would seem also from the finding of the fish bones in the old hernial sac that they must have escaped through some perforation of the appendix, of which the traces had been obliterated by the operation or in some other way, and that the inflammation and suppuration thus set up were the primary cause of the patient's symptoms.

A remarkable case which occurred in Prof. Billroth's surgical clinique is described by Dr. A. Wölfler in Langenbeck's 'Archives.'¹ It shows that besides adhesions, suppuration, and sloughing, the incarcerated appendix is also liable to other changes, which may give rise to symptoms resembling those of strangulation.

A youth, æt. 19, was admitted under Prof. Billroth in October, 1876, with a swelling the size of a hen's egg in his right groin. When first noticed two years before it was about the size of a walnut, and it had gradually increased since. He could readily return it, and he had not worn a truss. For two days the swelling had been red, hard, and painful, and he had been unable even with strong pressure to return it. At the same time he had suffered from nausea, vomiting, and constipation. He also related that four years before his admission he had been run over by a waggon, the wheels of which went over the lower part of his body. He had then for some time been unconscious, and had been kept from working for six weeks by what was thought to have been an attack of peritonitis on the right side. After taxis had been applied, the ordinary operation for herniotomy was determined upon. A sac was exposed and opened. About twenty drops of clear yellow fluid came away; no intestine was found, but there was what appeared to be another sac internal to the first one. After an unsuccessful attempt to reduce it by dividing the constrictions at the neck of the first sac, the second sac was also opened, and a cupful of yellowish watery fluid spurted forth. No intestine or omentum

¹ Langenbeck's 'Archiv für klinische Chirurgie,' vol. xxi, page 482.

was found. The interior of this second sac was of a red colour, and its surface was somewhat irregular. At first it was thought that it might be a part of the bladder, but this was easily disproved by passing a catheter and injecting milk into that viscus. Finally, it was ascertained by examination of a small portion of the wall of the sac under the microscope that it could only have been the dilated appendix cæci, part of which must have occupied the inguinal canal and part the interior of the abdominal cavity close to the internal ring. Severe peritonitis followed the operation, and there was also suppuration of the cyst, from which foul-smelling sloughs of connective tissue were discharged, but the patient finally recovered and left the hospital six weeks afterwards.

According to Dr. Wölfler the crush of the abdomen four years before had given rise to local peritonitis, and the appendix had thus become adherent to the abdominal parietes. At the same time a constriction had formed, which subsequently converted the appendix into a retention cyst.¹ Part of this cyst had finally projected into the inguinal canal, and a rapid increase of the contents had given rise to the symptoms of strangulation.

Judging from the cases which I have reported and quoted, I think we may gather the following facts with respect to the strangulation and incarceration of the appendix vermiformis :

1. The appendix vermiformis may form the sole contents of an inguinal or femoral hernia of the right side.²

2. When strangulated alone in the sac, it may give rise to all the symptoms of acute strangulation of the intestine, as in Case 4 and that related by Mr. Court, or on the other hand, as in Case 1 and that of the gentleman operated upon by Dieffenbach, the symptom of constipation may be absent.

¹ Dr. Wölfler refers to cases of cystic dilatation of the appendix which have occurred in the abdominal cavity, cf. Rokitsansky, 'Handbook of Path. Anatomy,' vol. iii, p. 184; and Virchow ('Krankhaften Geschwülste,' vol. i, p. 250) mentions one case in which the appendix was thus enlarged to the size of a big fist.

² It may even be found in a hernia of the other side, for Geissler mentions the case of a patient, seventy years old, who died of ascites, and in whom he found a much thickened vermiform process in an inguinal rupture of the *left* side. The cæcum lay in the middle of the abdomen and was very firmly attached to the small intestine and abdominal wall, cf. 'Jahresbericht' von Virchow, Gurlt, und Hirsch, vol. ii, 1867, p. 475.

8. If this strangulation is left unrelieved, acute inflammation with suppuration, sloughing, and perforation may follow, and this inflammation, as in Mr. Pick's case, may cause acute peritonitis and be fatal ; or, as in Mr. Durham's case, the patient may recover after operation with a faecal fistula.

4. The appendix may be incarcerated in an old hernia, and may become adherent to the walls of the sac. Here it may remain for many years without producing any serious symptom. It may then, as in Mr. De Morgan's case, become the site of ulceration from the lodgment of foreign bodies, and so set up suppuration in the hernial sac ; or, as in Case 2, it may be injured by taxis or a truss or by the pressure of portions of the bowel or omentum which have descended into the sac. Severe inflammation and great swelling may thus be set up in the process and the parts adjacent. Lastly, as in the singular case under Professor Billroth, some cystiform change may take place in the incarcerated appendix, and, by its great tension, set up all the symptoms of acute strangulation.

With respect to treatment, when we have to deal with small herniæ of the right side, whether inguinal or femoral, accompanied by vomiting, local pain, &c., it is well to bear in mind the possibility of the appendix being the only structure contained in the sac. We should then proceed to operate without delay, even though one of the chief signs of strangulation, such as constipation should be absent. If, on the other hand, we should have to operate upon a patient in whom from long incarceration the appendix has become adherent, Mr. Court's case shows that these adhesions may be sometimes divided, and the protrusion successfully returned. But when the appendix is much inflamed and suppurating as well as adherent, it is more difficult to decide upon the proper course to be pursued. If left in the sac it will probably be a source of continual irritation. Prolonged suppuration may follow, and even when this does not occur, it will interfere with the application of a truss, at the same time that it will keep open a passage for the descent of other portions of the intestine and omentum. I am therefore disposed to recommend that if it cannot safely be replaced within the abdomen, it should be ligatured and removed, as was done by myself in Case 2, and more successfully by Mr. Lucas in Case 4.

LIST OF SPECIMENS ADDED TO THE PATHOLOGICAL MUSEUM

DURING THE YEAR 1883-84.

By JAMES F. GOODHART, M.D.

THE pathological specimens added to the Museum during the past year are more numerous than usual, and many of them are by no means without interest. The list which is appended will almost be sufficient without additional remark, but I will shortly call attention to such of the additions as seem to be more particularly noteworthy either as rare examples of disease or as supplying a want of the Museum.

First may be noticed a specimen of syphilitic disease of the femur in an infant, 1000³⁰, presented by Mr. Robert W. Parker. And next two cases of spina bifida, 1002^{51, 52}, as being particularly valuable because they are well displayed by dissection. For the dissection of the one we are indebted to Mr. John Poland; for the other to Dr. Anningson, of Cambridge, who undertook it at the request of Professor Humphry and for the purpose of a more detailed examination.

1129⁵¹ and ⁵² are two interesting pelves from Dr. Hale White and Mr. Lane, both intended to illustrate some of the points in the pathology of Charcot's disease. 1069⁴⁰ and 1132⁵¹ *a* (both from the same case) are remarkable specimens because they appear to be allied to the disease now called osteitis deformans, after Sir James Paget's description, and

They are particularly interesting because they still further illustrate the tendency of this peculiar disease not only to produce bone in enormous quantity after the manner of a normal development but also to be associated with the growth of other osseous tumours. In this case, however, the growth is entirely a new one and not a cancer. 1117⁸, a dissection of a bone removed by Mr. John Parnell, is another specimen which is a distinct addition to the treasures of the Museum. It is an interesting specimen of injury at the elbow-joint, and illustrates a case of suppurative arthritis in early infancy—a specimen it which was added to the Museum last year by Mr. Carter. It is an illustration of a disease to which but little attention has yet been directed. Suppurative arthritis is usually regarded as being essentially considered and distinguished from the suppurative processes which occur in congenital syphilis and other conditions. 1118⁸ and 1315⁸ are instructive examples of disease of the hip in the adult periods of life.

Amongst the diseases of the heart there are some very instructive specimens. 1314⁸, dilatation of both sides of the heart from acute rheumatism. 1315⁸, dilatation of the left ventricle in rheumatism. 1400⁸, thrombosis of the coronary artery and rupture of the heart, and 1411⁸ a specimen of friction ulcer on the mitral valve.

1316⁸ is a specimen of disease which may be examined in connection with a specimen placed on the shelves last year. Both are examples of the different causes of great dilatation of the pulmonary artery, and both possess features of physiological, chronic, and pathological interest which will repay the time devoted to their examination. 1450⁸ is a dried and injected specimen of dilatation of the aorta beyond the left subclavian artery. This is a very valuable specimen and we are indebted to Dr. John White for its preservation and dissection.

Amongst diseases of the nervous system attention may be called to a case of syringomyelia, 1592⁸, by Dr. Frederick Fox et al. to a tumour of the thalamus by Dr. Pye-Smith, and to a neuro-vascular tumour about the left Casserian ganglion (probably an illustration of a distinct group of cases) by Dr. Meyer.

1593⁸ is an enormous bony growth from the head of an old man, removed and presented by Mr. Etches. 1657¹¹, a fleshy

ulcer on the back by Dr. Taylor, is a similar preparation to one already presented last year by Mr. Davies-Colley, 1657¹⁰. Both together illustrate a disease which may be called a new one, so little is it yet part of the literature of medicine. Mr. Durham has described other cases of a like character.

Mr. Jacobson contributes a case of cancer of the tongue in the site of an old syphilitic ulcer, 1670⁶⁶, and the Curator a specimen of lingual cyst, rare as a Museum specimen, though not uncommon in practice.

In the additions to the lung and œsophagus series, there are several specimens which suggest practical points relating to symptoms and treatment, but none perhaps that require special mention.

1801¹⁰ is a very rare specimen of gastric ulcer in a newborn infant, presented by Mr. Cock. 1854⁶³ is a specimen, also rare, of double stricture of the colon, by Sir Wm. Gull.

Amongst diseases of the liver are specimens of cystic liver, by Dr. Hale White, and interesting examples of lympho-sarcomatous growth, by Dr. Wilks and Dr. Pye-Smith; but perhaps most interesting of all in the series is 1922⁸¹. Several authors, Frerichs, Perls, and the late Dr. Hilton Fagge particularly, have described a form of cancer which so much resembles cirrhosis as to be likely to be mistaken for it, and at first sight this was supposed to be a disease of that kind; the liver has all the appearance of extreme cirrhosis, and scattered throughout it are circular masses of soft material which exactly resemble a growth of cancer. The nature of the nodules had indeed appeared to me to be so certain that I had not examined them further, until it was pointed out to me by Dr. Pitt that the masses of soft material appeared to him, after microscopical examination, to be simply masses of atrophic liver-cells. Since then I have examined the nodules and quite agree that this is the case, and the disease is therefore cirrhosis simulating cancer, a rare and I think hitherto undescribed condition.

In the genito-urinary series there are interesting specimens presented by Mr. Farrant Fry; a dermoid polypus from the bladder, a specimen of great rarity, by Mr. Bryant; a sarcoma of an undescended testis, by Dr. Moxon, &c.

In this short enumeration the interesting specimens are by no means exhausted. Let them serve as an advertisement

of the 130 new preparations which form a part, and only a small part, of the work of our Museum Superintendent, Mr. Betts, and his assistant, during the past year. The labour, on their part, has been unsparing, it will be best requited by the intelligent appreciation which it will certainly receive.

- | <i>No.</i> | <i>Donor.</i> |
|--|------------------------------|
| 1000 ¹⁶ . Atrophy of the Bones of an Upper Extremity, after Hemiplegia. | <i>Dr. Hale White.</i> |
| 1000 ²⁰ . Syphilitic Disease of the Femur in an Infant. | <i>Mr. Robert W. Parker.</i> |
| 1002 ⁵¹ . A Spina Bifida dissected. The dissection by Mr. John Poland. | <i>Mr. Bryant.</i> |
| 1002 ⁵² . A Spina Bifida dissected. The dissection by Dr. Annington, of Cambridge. | <i>Mr. Lucas.</i> |
| 1011 ¹⁰ . The Cervical Spine with the Foramen Magnum, being the repair of what appears to have been very extensive disease. The man died from some disease unconnected with the specimen. | <i>Dr. Goodhart.</i> |
| 1030. Dislocation forwards of the 6th Cervical Vertebra; the injury occurred six and a half months before death, but the evidences of attempted repair are still but meagre. | <i>Mr. Durham.</i> |
| 1030 ¹⁰ . Section of the Cervical Spine in which a Fracture of the 4th and 5th, and Displacement of the 5th from the 6th Vertebra have undergone repair. | <i>Mr. Bryant.</i> |
| 1043 ⁴⁶ . Repaired Fracture of the Sternum. | <i>Mr. Arbuthnot Lane.</i> |
| 1043 ⁵⁶ . Fracture of Greater Cornu of Hyoid Bone. | <i>Mr. Arbuthnot Lane.</i> |
| 1050 ⁹⁰ . Hæmaturia beneath the Pleuræ from Fracture of the Ribs. | <i>Mr. Howse.</i> |
| 1051 ²⁶ . Specimens of Fractured Ribs from a Man, aged 71, suffering from Senile Dementia. | <i>Dr. Paddison.</i> |
| 1055 ⁶⁰ . Longitudinal Section of the Calvaria of an Infant with the Occipital Bone displaced backwards by what appears to be an abnormal fleshy growth at the hinder part of the Falx Cerebri. | <i>Dr. Hale White.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|---|----------------------------|
| 1069 ⁴⁰ . Osseous Tumour of the Right Temporal Region of Skull. The centre of the tumour is occupied by a softer material, but felt unlike an ordinary sarcomatous tumour. The right tibia and femur showed chronic ostitic changes, such as are seen in cases of Osteitis Deformans, but the bones were neither lengthened nor curved (1132 ⁵¹ <i>a</i>). | <i>Dr. Pavy.</i> |
| 1081 ¹⁵ . Sarcoma of Temporal Bone from the same patient as 1162 ⁷⁰ . | <i>Mr. Durham.</i> |
| 1081 ⁹⁵ . Calvaria showing upon its exterior large Cancerous Erosions of the Bone, and on its inner aspect three large protruding masses of growth corresponding. The Adrenals, Liver and Skin were also affected. From a Child aged six months. | <i>Dr. Pye-Smith.</i> |
| 1085 ⁷⁶ . Portion of Calvaria showing a detached fragment of the internal table glued in position by blood-clot and lymph. | |
| 1110 ⁸⁷ . Fibrous Ankylosis of a Fractured Humerus. | <i>Mr. Arbuthnot Lane.</i> |
| 1117 ⁴² . Vertical Section through the Radius and Bones of the Wrist, with the Muscles dissected to show the condition of parts in separation of the Lower Epiphysis of the Radius. | <i>Mr. John Poland.</i> |
| 1124 ⁴⁷ . Sebaceous Cyst removed from the palmar surface of the terminal phalanx of the left ring finger. | <i>Mr. John Poland.</i> |
| 1129 ²¹ . Pelvis from a case of Charcot's Disease. | <i>Dr. Hale White.</i> |
| 1129 ²² . A Pelvis from a case of Charcot's Disease with Spondylolisthesis. | <i>Mr. Lane.</i> |
| 1132 ⁵¹ <i>a</i> . Chronic Ostitis of Femur and Tibia ? Osteitis Deformans. | <i>Dr. Pavy.</i> |
| 1158 ¹² . A Section of a Femur. Amputation had been performed fifty-three days before death. The medulla in several parts is converted into deposits of dirty-looking pus. | <i>Mr. Bryant.</i> |
| 1158 ⁴⁰ . A Section of a Stump in which the Patella was pegged to the Femur ten days before death. | <i>Mr. Golding-Bird.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|---|----------------------------|
| 1162 ⁷⁰ . An enormous Osteo-Sarcomatous Tumour from the thigh of a Boy aged nine years. | <i>Mr. Durham.</i> |
| 1284 ⁸² . Monstrous toes removed by Operation. | <i>Mr. Davies-Colley.</i> |
| 1306 ¹⁰ . Old Partial Dislocation inwards of the Radius and Ulna, and Detachment, and Outward Rotation of the outer half of the Epiphysis of the Humerus. | <i>Mr. Bryant.</i> |
| 1315 ⁵⁶ . Suppurative Arthritis from Epiphysial Disease in an Infant of eight months old. | <i>Mr. John Poland.</i> |
| 1317 ¹¹ . Parts removed in Excision of the Hip. The neck of the bone has all but disappeared, and the head of the bone, of a much earlier age than that which should correspond with the trochanteric portion of the specimen, was found lying in the cavity of the hip-joint. From a Child of five years. | <i>Mr. Howse.</i> |
| 1318 ⁷² . A Section of a Hip-Joint showing very partial Ankylosis. The patient, a man of sixty-eight, died from Cancer independent of his hip which had been stiff for years. He was under Mr. Hilton in 1859 for hip disease. | <i>Mr. Howse.</i> |
| 1329 ⁵⁹ . A Knee-Joint eight years after Excision. | <i>Mr. J. Farrant Fry.</i> |
| 1329 ⁶⁰ . A Knee-Joint some months after Excision. | <i>Mr. Howse.</i> |
| 1329 ⁶¹ . The parts removed in Excision of the Knee-Joint. The disease originated in Hydatid Disease, some of the small cysts of which may still be seen in the cavity of the internal condyle of the femur on the posterior aspect of the preparation. | <i>Mr. Howse.</i> |
| 1394 ²⁷ . Dilatation of both sides of the Heart from a case of Acute Nephritis. | <i>Dr. Wilks.</i> |
| 1394 ²⁸ . Dilatation of the Left Ventricle in Diphtheria. | <i>Dr. Wilks.</i> |
| 1396 ³⁷ . A Heart which has undergone extensive fibroid changes. The Endocardium behind the mitral valve is converted into a thick, opaque tough layer, and the wall of the Ventricle is dilated into an aneurismal pouch. | <i>Dr. Mahomed.</i> |
| 1400 ⁷⁷ . Three Horizontal Sections of the Left Ventricle, to | |

No.

Donor.

show a fibrous appearance in the wall. The coronary arteries were diseased and thrombosed and in the recent state it appeared that this had induced a state of infarction of the muscle, and that this had led first to aneurismal dilatation of the part and then to rupture of the wall, from which the patient died.

Dr. Wilks.

1401²⁷. Friction or Inoculation Ulcer on the Ventricular Flap of the Mitral Valve.

Dr. Wilks.

1450⁴². The base of the Heart with the Vessels showing Congenital Malformation of the Pulmonary Valves, and a greatly dilated Pulmonary Artery.

Dr. Hilton Fagge.

1450⁶⁶. Aneurism upon a Branch of the Pulmonary Artery.

Dr. Goodhart.

1450⁸⁸. A dried dissected Specimen of Coarctation of the Aorta.

Dr. Hale White.

1451. The Arch of an Aorta with a ragged communication between it and the Œsophagus an inch beyond the Left Subclavian Artery. This was produced by a small fish bone which was swallowed.

1451¹. The Fish Bone attaching to the preceding preparation.

Surgeon-Major Hume Spry.

1501¹². The left half of the Larynx and adjacent parts to show a cancerous state of the Tonsil, Tongue, and Cervical Glands, and a Ligature which was placed on the External Carotid for Hæmorrhage. The patient lived only about twenty-four hours after the operation.

Mr. Davies-Colley.

1501⁸⁸. Part of a Splenic Artery showing the mode of restoration of the channel after Embolism by shrinking of the clot, and a process of Veneering.

Dr. Goodhart.

1541⁴⁹. Mediastinal Sarcoma from a Child.

Mr. Briscoe.

1542¹⁰. A large mass of Carcinomatous Lymphatic Glands.

Mr. Durham.

1562⁹¹. Sections of Spinal Cord showing the irregular dilatation in the posterior segment known as Syringomyelus.

Dr. Taylor.

- | <i>No.</i> | <i>Donor.</i> |
|---|----------------------------|
| 1565 ⁹³ . Cerebral Abscess of three weeks' date after Injury in a Child of a year and three quarters. | <i>Mr. Durham.</i> |
| 1575 ⁴⁶ . Yellow Tubercular Tumour of the Right Thalamus. | <i>Dr. Pye-Smith.</i> |
| 1575 ⁵⁰ . Portions of Brain containing numerous hæmorrhagic extravasations (? Lymphomata). From a case of Acute Hodgkin's Disease. | <i>Dr. Mahomed.</i> |
| 1576. A Section of the Brain and its coverings, showing a large tumour in the frontal region. It had extended through the skull and formed a localised tumour under the scalp—and exploratory trephinations had been performed, in the hope, in ignorance of its extent, of a possible enucleation. | <i>Dr. Moxon.</i> |
| 1576 ⁵² . A Fibro-Sarcomatous Tumour connected with the Membranes at the base of the Brain and involving the Left Casserian Ganglion. | <i>Dr. Moxon.</i> |
| 1576 ⁶¹ . Gliomatous Tumour of the Cerebellum. | <i>Dr. Moxon.</i> |
| 1576 ⁶² . A Gliomatous Tumour involving the anterior part of the fornix and spreading into the ventricle on the left side and into the white matter of the cerebral hemisphere on the right. | <i>Dr. Herbert Illott.</i> |
| 1590 ¹⁶ . A Section of the Foramen Magnum of a Child's Skull and of the upper Cervical Vertebræ, with the membranes of the spinal cord <i>in situ</i> , showing the condition of part in Chronic Basal Meningitis. A number of membranous adhesions are seen about the upper part of the head. These had led to chronic hydrocephalus. | <i>Dr. Goodhart.</i> |
| 1601 ¹⁵ . Carcinoma of Dura Mater and Skull. Many other parts of the body were affected. | <i>Dr. Taylor.</i> |
| 1612 ⁶¹ . Tumour of Pituitary Body. | <i>Dr. Taylor.</i> |
| 1635 ⁸¹ . Molluscum Fibrosum. | <i>Dr. Taylor.</i> |
| 1637 ¹⁰ . Section of Foot with a Chronic Ulcer of Epitheliomatous nature on the heel—the disease has invaded the os calcis. | <i>Mr. Bryant.</i> |
| 1652 ¹⁷ . A Horny Growth from the Head of an Old Man of eighty-one. It was removed all but the last quarter of an inch, which continued to grow. | <i>Mr. Etches.</i> |
| 1657 ¹¹ . Lympho-Sarcomatous ulceration of Back. | <i>Dr. Taylor.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|---|------------------------|
| 1670 ⁶¹ . Enlargement of the Papillæ of the Tongue. | <i>Mr. Jacobson.</i> |
| 1670 ⁶¹ . Ichthyosis of the Tongue. | <i>Dr. Moxon.</i> |
| 1670 ⁶⁵ . A Cancerous Ulcer on the right side of the Tongue which commenced in the site of a Chronic Ulcer ; some slight Ichthyotic condition surrounding it. | <i>Mr. Jacobson.</i> |
| 1673 ³⁰ . A Cyst at the base of the Tongue. | <i>Dr. Goodhart.</i> |
| 1674 ⁵ . Cancerous Ulceration of the Root of the Tongue and Epiglottis. | <i>Dr. Goodhart.</i> |
| 1674 ⁶ . Epithelioma of the Tonsil extending to the Epiglottis. Death from hæmorrhage. | <i>Mr. Symonds.</i> |
| 1690 ⁷⁵ . A Child's Larynx containing large masses of Warts upon the Left Vocal Cord, and in the middle line immediately above the opening made by Tracheotomy. | <i>Dr. Taylor.</i> |
| 1697 ⁷ . Syphilitic Ulceration of Larynx with scarring of the Trachea and Stenosis of the Bronchus. | <i>Dr. Taylor.</i> |
| 1697 ¹¹ . Ulceration of the Larynx with associated disease of Skin and Testes of uncertain nature (with 1657 ¹¹). | <i>Dr. Taylor.</i> |
| 1716 ¹⁹ . Tracheitis and Ulceration excited by the presence of an India-rubber Tracheotomy tube. | <i>Dr. Pye-Smith.</i> |
| 1718 ⁴³ . The Apex of the Right Lung showing a thick mass which looks like a much-thickened pleura, but which contains largely dilated and sacculated bronchial tubes, proving that it is in great measure obsolete lung tissue. | <i>Dr. Carrington.</i> |
| 1718 ⁴⁵ . Sections of the Lung invaded by a large mass of cancer from the Mediastinum. The preparation is mounted to show the extreme dilatation of the bronchial tubes which has resulted therefrom. | <i>Dr. Pavy.</i> |
| 1718 ⁵⁷ . Saccular Dilatation of the Bronchial Tubes. | <i>Dr. Mahomed.</i> |
| 1718 ⁶⁸ . Syphilitic Stenosis of Bronchus. | <i>Dr. Moxon.</i> |
| 1725 ⁴⁹ . The greater part of the Lower Lobe of the Right Lung solidified by infarction. | <i>Dr. Pavy.</i> |
| 1738 ⁶¹ . Extensive Cavitation of the base of the Left Lung from the extension of a spinal abscess. | <i>Dr. Moxon.</i> |

454 *List of Specimens added to the Pathological*

- | <i>No.</i> | <i>Donor.</i> |
|---|--------------------------------------|
| 1743 ⁷¹ . Fibroid Disease of the Lung with Emphysema and Thrombosis of the Pulmonary Artery. | <i>Dr. Moxon.</i> |
| 1748 ⁵ . Secondary Sarcoma of the Lung. | <i>Dr. Pye-Smith.</i> |
| 1766 ⁵⁷ . Portion of Lung with the visceral layer of the Pleura, showing semi-cartilaginous linear thickenings occupying the position of the intercostal spaces and small disseminated fibroid nodules of similar material. The lung, is very generally emphysematous. | <i>Dr. Goodhart.</i> |
| 1766 ⁷⁷ . Part of a Pleura Costalis to show numerous ulcers upon its surface. From a case of empyema with gangrene of the lung. | <i>Dr. Wilks.</i> |
| 1783 ⁷¹ . An Ear of Grass discharged from an Abscess in the side; it was probably swallowed and drawn into the lung, and thus worked its way to the surface. | <i>Dr. Wilks.</i> |
| 1789 ³⁷ . Œsophageal Pouch. | |
| 1789 ³⁸ . An Œsophageal Pouch. | <i>Mr. Jacobson.</i> |
| 1791. Early Cancerous Ulceration and Stricture of the Œsophagus. | <i>Dr. Mahomed.</i> |
| 1796 ⁵ . Hour Glass Contraction of Stomach. | <i>Dr. Carrington.</i> |
| 1796 ⁶ . Hour Glass Contraction of Stomach. | <i>Dr. Hale White.</i> |
| 1799 ³⁵ <i>a</i> . Portions of Œsophagus and Intestine from a case of poisoning by Chloride of Zinc. | <i>Dr. Stevenson and Mr. Crosse.</i> |
| 1801 ¹⁰ . The Stomach of a newly-born Infant everted and showing to the left of the preparation a small deep ulcer with raised edges which led to fatal hæmorrhage. | <i>Mr. Cock.</i> |
| 1816 ¹⁶ . Stomach eight months after Gastrostomy. | <i>Mr. Howse.</i> |
| 1829 ⁸¹ . The first part of the Duodenum with the pylorus to the right. A large chronic ulcer partially healed is seen on its opposite face. The Arteria Pancreatico Duodenalis has been opened by the disease and fatal hæmorrhage has resulted. A blue glass rod indicates the position of the vessel. | <i>Mr. Golding-Bird.</i> |
| 1831 ³² . Intestinal Anthrax. | <i>Dr. Mahomed.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|--|---------------------------|
| 1831 ²³ . Intestinal Anthrax. | <i>Mr. John Poland.</i> |
| 1831 ²⁴ . Gastric Anthrax. | <i>Mr. John Poland.</i> |
| 1832 ⁵¹ . A piece of Small Intestine dilated and thickened, with an opening through it to the surface of the abdominal wall. From a patient admitted for intestinal obstruction (the same case as 1887 ⁷⁶). Littré's operation was performed some time before death and the symptoms were for a time ameliorated. | <i>Mr. Symonds.</i> |
| 1832 ⁵⁶ . Portions of Intestine showing an ulcerated and undermined condition of the Mucous Membrane, apparently from the burrowing inwards of an external abscess. | <i>Mr. Davies-Colley.</i> |
| 1845 ²⁶ . Part of the Small Intestine presenting extreme tubercular disease of all the coats. | <i>Dr. Taylor.</i> |
| 1846 ⁴² . A piece of Small Intestine abnormally convoluted by chronic inflammation and thickening of the Mesentery, a condition which might very readily have given rise to Ileus. | <i>Mr. Symonds.</i> |
| 1849 ⁶⁴ . A Vertical Section of an Intussusception. | <i>Dr. Moxon.</i> |
| 1851 ⁹⁶ . Acute Thrombosis of the Superior Mesenteric Vein. | <i>Dr. Hilton Fagge.</i> |
| 1854 ⁶² . Double Stricture of the Transverse Colon. | <i>Sir W. Gull.</i> |
| 1863 ⁶⁰ . A portion of Large Intestine, the mucous membrane of which is thickly covered with small circular ulcers. The patient suffered from dysentery and died with hepatic and cerebral abscess. | <i>Dr. Pavy.</i> |
| 1881 ⁹⁰ . Cancerous stricture of Splenic Flexure, with two elongated tags or polypi growing from its lower aspect. | <i>Dr. Moxon.</i> |
| 1884 ¹ . A lobulated Epithelioma of the sigmoid flexure shown in a series of transverse sections. | <i>Dr. Pavy.</i> |
| 1886 ³¹ . The lower part of the Rectum and adjacent parts seen in vertical and transverse section. The bowel is greatly contracted, as seen by the transverse section above, a blue glass rod having been passed along it. The trabeculated and fistulous state of parts is shown below. | <i>Mr. Bryant.</i> |
| 1887 ⁶⁶ . A Globular Rectal Polypus. | <i>Dr. Mahomed.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|---|--------------------------|
| 1887 ³ . The growth removed at, and the condition of the parts remaining after, an operation for the extirpation of a growth in the colon, which had caused intussusception. The intestine has been hardened and cut vertically. The patient lived but a few hours after the operation (1832 ⁵¹). | <i>Mr. Symonds.</i> |
| 1900. Hepatic Abscess. | <i>Dr. Taylor.</i> |
| 1909 ⁴ . Cystic Disease of the Liver. | <i>Dr. Hale White.</i> |
| 1909 ⁵ . Cysts in the Liver, produced by decomposition and the formation of gas in the tissue. | <i>Dr. Hale White.</i> |
| 1914 ⁶ . Cirrhosis of the Liver of a Child. | <i>Dr. Goodhart.</i> |
| 1921 ⁷ . A Section of the Liver infiltrated with a firm white lymphomatous growth. The patient died from Hodgkin's Disease. | <i>Dr. Pye-Smith.</i> |
| 1921 ⁸ . Lympho-Sarcoma of Liver. | <i>Dr. Wilks.</i> |
| 1922 ⁹ . Cirrhosis of the Liver simulating Cancer. The liver has all the external features of cirrhosis, but its section is studded with globular masses of various size, soft, discoloured by blood, and in all respects like a cancerous growth. A microscopic examination showed that these were masses of liver-cells in process of atrophy encircled by the fibroid material of a cirrhosis. | <i>Dr. Pye-Smith.</i> |
| 1922 ¹⁰ . A Cirrhotic and Cancerous Liver. | <i>Dr. Hilton Fagge.</i> |
| 1961 ¹¹ . Part of the Liver with the Gall-Bladder—portal fissure and duodenum <i>in situ</i> . The gall-bladder is closely contracted upon some gall-stones, one of which has caused ulceration of the colon (at the upper part of the preparation), and the common duct is greatly dilated and blocked by a large gall-stone near its termination in the duodenum (seen in the lower part of the specimen). | <i>Dr. Taylor.</i> |
| 1964 ¹² . Cancer of the Liver secondary to Cancer of the Neck of the Gall-Bladder. | <i>Dr. Pavy.</i> |
| 1996 ¹³ . Chronic Enlargement of the Spleen from a Rickety and possibly syphilitic Child of fifteen months old. | <i>Dr. Goodhart.</i> |
| 1996 ¹⁴ . Chronic Enlargement of the Spleen from a Child. | <i>Dr. Goodhart.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|---|----------------------------|
| 2010 ⁶⁵ . Part of a Spleen with a Cyst beneath its Capsule. | <i>Dr. Wilks.</i> |
| 2014 ²⁵ . An Atrophied Spleen, weighing less than an ounce, with thickened Capsule, from a Man aged forty-one, who died of Chronic Phthisis. | <i>Dr. Mahomed.</i> |
| 2021 ³⁶ . Sarcoma of the Adrenals from a Child five months old. | <i>Dr. Pye-Smith.</i> |
| 2044 ³⁰ . Cystic Kidney in a Child a month old. | <i>Mr. Howse.</i> |
| 2077 ⁶⁰ . A Calculus from the Pelvis of the Kidney, consisting almost entirely of triple phosphate of magnesium and ammonium. | <i>Dr. Hale White.</i> |
| 2078 ¹ . A Kidney with two Ureters, one greatly dilated and opening into a Cyst in the floor of the Urethra. | <i>Dr. Moxon.</i> |
| 2078 ² . The Bladder, lower part of Rectum, and Kidneys of a Child. The left kidney is cystic and obsolete. The right is large and its pelvis and ureter dilated, the latter opening into a passage communicating with the urethra and rectum in the prostatic region. | <i>Mr. J. Farrant Fry.</i> |
| 2102 ¹¹ . Early Scrofulous Ulceration of the Bladder in a Boy of ten years. | <i>Mr. Symonds.</i> |
| 2102 ¹² . A Bladder, the mucous membrane of which has upon it three superficial scrofulous ulcers. The prostate and left ureter are much diseased. | <i>Dr. Wilks.</i> |
| 2104 ²⁶ . Bladder entirely full of a soft cancerous growth. | <i>Mr. J. Farrant Fry.</i> |
| 2104 ³¹ . A Dermoid Polypus, with hair growing from its surface, removed from the bladder. | <i>Mr. Bryant.</i> |
| 2104 ³² . Calculi removed from the bladder of the preceding case. | <i>Mr. Bryant.</i> |
| 2339 ⁷¹ . Sarcoma of an Undescended Testis. | <i>Dr. Moxon.</i> |
| 2458 ³¹ . Half of a much-thickened Omentum which is full of Tubercle. | <i>Dr. Moxon.</i> |
| 2470 ³¹ . A Stomach and Great Omentum infiltrated with Colloid Cancer; to the right of the preparation is the sac of the lesser omentum laid open; it contained fluid in the recent state. | <i>Dr. Pye-Smith.</i> |

- | <i>No.</i> | <i>Donor.</i> |
|---|--------------------------|
| 2470 ³² . A part of the Peritoneal Aspect of the Diaphragm from the same case as the preceding specimen. | <i>Dr. Pye-Smith.</i> |
| 2498 ⁶⁵ . Hernia by rupture of the anterior wall of the Inguinal Canal. The preparation has been dissected by Mr. John Poland. | <i>Mr. Golding-Bird.</i> |
| 2525 ⁴¹ . A Uterus with a Foetus enveloped in its Placenta. It had the appearance of having been some time dead. | <i>Dr. Beale.</i> |

STATISTICAL SUMMARY
OF THE
PATIENTS TREATED IN GUY'S HOSPITAL DURING THE
YEAR 1883.
By J. C. STEELE, M.D.

Patients in hospital 1st January, 1883	440
Admitted during the year	4681
Discharged as cured	1894
Relieved	2010
Unrelieved	317
Died	487
Remaining 31st December, 1883	413
Average number daily resident	446
Mean residence of each in days	34·49
Rate of mortality per cent.	10·34

MEDICAL WARDS.

Patients in hospital 1st January, 1883	150
Admitted in the course of the year	1728
Discharged as cured	444
Relieved	822
Unrelieved	129
Died	327
Remaining 31st December, 1883	156
Average number resident daily	149
Mean residence of each in days	31·39
Rate of mortality per cent.	18·97

SURGICAL WARDS.

Patients in hospital 1st January, 1883	290
Admitted in the course of the year	2953
Discharged as cured	1450
Relieved	1188
Unrelieved	188
Died	160
Remaining 31st December, 1883	257
Average number resident daily	297
Mean residence of each in days	36·29
Rate of mortality per cent.	5·35

Summary distinguishing the Sexes with the relative Mortality.

MEDICAL WARDS.						SURGICAL WARDS.					
Admitted.		Discharged.		Died.		Admitted.		Discharged.		Died.	
M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
988	740	783	612	205	122	1931	1022	1841	985	116	44

Retrospective Summary of In- and Out-Patients who have received Relief at the Hospital during the past ten years.

	1874.	1875.	1876.	1877.	1878.	1879.	1880.	1881.	1882.	1883.
IN-PATIENTS.										
Under treatment during the year	5,776	5,854	5,722	5,544	5,710	5,727	5,189	4,923	5,150	5,121
Discharged cured	1,354	854	1,511	1,550	1,551	1,723	1,820	1,737	1,759	2,010
Relieved	2,925	3,452	2,664	2,493	2,695	2,469	2,060	1,957	2,242	1,894
Unrelieved	334	419	435	460	371	418	399	329	258	317
Died	594	560	593	498	555	545	477	465	451	487
Rate of mortality per cent.	11.40	10.60	11.39	9.95	10.72	10.55	10.24	10.36	9.57	10.34
Mean residence of each in days	38.6	37.3	39.5	39.5	37.9	39.2	39.5	36.7	34.4	34.4
Number of accidents	852	928	871	803	824	935	869	994	997	940
Deaths from accidents	102	95	83	81	76	77	73	78	72	59
OUT-PATIENTS.										
Surgical cases	3,801	3,313	3,505	3,988	3,919	3,634	3,928	3,195	3,661	4,177
Medical cases	2,919	2,987	3,265	3,041	2,838	2,884	2,773	2,843	2,703	2,685
Diseases of the eyes	3,083	2,717	3,252	3,028	3,059	2,652	2,654	2,286	2,502	2,613
Diseases peculiar to women	1,644	1,745	1,902	2,114	1,963	1,416	1,540	1,277	1,400	1,595
Diseases of the skin	989	1,170	985	1,000	1,031	963	947	923	1,013	989
Diseases of the ear	1,089	990	1,251	1,318	1,189	1,060	1,072	939	1,107	1,308
Casual or minor medical cases.	7,042	7,143	7,013	7,535	8,107	7,863	9,056	11,810	12,854	11,287
Casual or minor surgical cases.	43,573	41,363	38,649	35,247	21,934	17,781	20,818	23,670	26,046	16,636
Tooth extractions	3,758	2,278	2,783	2,801	3,261	3,695	2,850	2,320	1,346	2,012
Minor accidents	14,746	9,764	11,005	11,449	11,353	11,020	12,173	13,170	12,909	12,907
Women confined at their homes	2,449	2,334	2,449	2,540	2,552	2,613	2,593	2,483	2,504	2,637
Deaths after confinement	17	9	13	9	5	3	8	6	7	10

L I S T
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1882.

University of Oxford.

Final Examination for the Degree of Bachelor of Medicine.

J. A. P. Price.

First Examination for the Degree of Bachelor of Medicine.

R. Moody Ward.

University of Cambridge.

Final Examination for the Degree of Bachelor of Medicine.

J. E. Viney.

Second Examination for the Degree of Bachelor of Medicine.

S. H. Agar.

J. T. J. Morrison.

F. R. B. Bisshopp.

Examination for the Degree of Bachelor of Surgery.

G. N. Pitt.

University of London.

Examination for the Degree of Doctor of Medicine.

H. Davy.

E. Penny.

E. H. Paddison.

B. N. Rake.

G. H. Russell.

Final Examination for the Degree of Bachelor of Medicine.

First Division.

E. L. Adeney.

Obtained First-Class Honours in Medicine and Honours in Forensic Medicine.

462 *Gentlemen admitted to Degrees, &c., in the year 1882.*

O. J. Currie.

Obtained First-Class Honours in Forensic Medicine.

W. E. Fielden.

Obtained Honours in Medicine.

L. E. Shaw.

Obtained Honours in Medicine.

L. C. Wooldridge.

Obtained the Gold Medal in Medicine and First-Class Honours in Forensic Medicine.

W. R. Dakin.

|

B. Parry.

Intermediate Examination in Medicine.

First Division.

G. E. C. Anderson.

Obtained the Exhibition and Gold Medal in Anatomy, First-Class Honours in Organic Chemistry, and Honours in Physiology and Histology.

W. H. Bowes.

Obtained Honours in Physiology and Histology.

C. S. Spong, B.Sc.¹

Obtained First-Class Honours in Organic Chemistry and Honours in Physiology and Histology.

W. I. Watson.

Obtained Honours in Anatomy.

Second Division.

H. C. E. Cooper.

Obtained Honours in Anatomy and in Physiology and Histology.

R. M. H. Randell.

Obtained Honours in Materia Medica and in Physiology and Histology.

J. J. D. Vernon.

Obtained Honours in Physiology and Histology.

R. W. Brogden.

C. Caldecott.

|

J. H. Sellick.

A. E. Taylor.

Physiology only.

O. Gross.

Preliminary Scientific (M.B.) Examination.

First Division.

H. E. Crook.

Obtained Honours in Organic Chemistry.

W. H. W. Elliot.

Obtained Honours in Botany.

Gentlemen admitted to Degrees, &c., in the year 1882. 463

H. J. Blackler
E. H. Brock.
W. F. Clarke.

H. Smith.

E. R. Davies.
H. K. Roper.
C. S. Simpson.

Second Division.

L. Bidwell.
A. T. Brown.
F. F. Burghard.
H. E. Craig.
S. F. Holloway.

T. Fisher.
S. E. Prall.
W. N. Ridsen.
A. Scott.
O. Legg.

Final Examination for the Degree of Bachelor of Surgery.

Second Division.

W. R. Dakin.

Final Examination for the Degree of Bachelor of Science.

F. Lever.

Obtained Honours in Physiology.

G. E. Halstead.

F. B. W. Phillips.

Unibersity of Durham.

First Examination for the Degree of Bachelor of Medicine.

H. J. Hillstead.
A. E. Larking.

E. W. Simmons.
W. L. Blight.

Unibersity of Edinburgh.

Doctor of Medicine.

A. G. Barrs.

Unibersity of Aberdeen.

Doctor of Medicine.

H. J. Liebsstein.

***Final Examination for the Degree of Bachelor of Medicine and
Master of Surgery.***

J. W. Hodgson.

H. A. Phillips.

Royal Unibersity of Ireland.

Examination for Degree of Doctor of Medicine.

Upper Pass Division.

J. M. Prendergast.

Examination for Degree of Master of Surgery.

J. M. Prendergast.

Diploma in Obstetrics.

J. M. Prendergast.

464 Gentlemen admitted to Practice, &c., in the year 1882.

British Medical Service.

London Examination in February.

S. O. Stuart, 1940 marks.

London Examination in August.

J. M. Prendergast, 1935 marks.

Netley Examination in August.

S. O. Stuart.

Royal College of Physicians.

Examination for the Membership.

A. H. Jones, M.D.

Final Examination for the Licence.

T. M. Day.	J. M. Owen.	R. F. Cox.
C. J. Harper.	R. H. Perks.	W. C. Dendy.
R. Parry, M.B.	A. Scott.	W. H. W. Strachan.
G. B. Prabhakar.	L. Stokes.	J. E. Viney.
E. H. Booth, M.B.	H. Blatherwick.	H. C. Dixon.
J. Cock.		

First Examination for the Licence.

March.

J. Cock.	E. Sharpley.	T. H. Miller.
H. Lamb.	L. Stokes.	A. C. Deare.
G. Kendall.	J. G. Milnes.	

May.

H. E. Counsell.	W. H. Brenton.	C. E. Bean.
J. Dowson.	F. C. Payne.	T. B. Winter.
H. Blatherwick.	W. H. W. Strachan.	F. N. Shillingford.
W. C. Dendy.	H. H. Wright.	

August.

E. W. Du Buisson.	R. Creasy.	A. Meyrick-Jones.
J. Crisp.	H. Howard.	J. C. Underwood.
S. H. Seccombe.	F. W. Langridge.	E. Deane.
S. Wachter.	S. W. Owen.	A. E. Wilson.
E. A. Farr.	R. J. Cook.	C. G. Wallis.
G. F. E. Morgan.	G. H. Hugill.	

November.

L. Burroughs.	C. J. Fuller.	G. G. O. Phillips.
W. A. Cahill.	J. H. Harris.	J. N. Phillips.
A. Z. C. Oressy.	J. D. Howe.	H. E. Rowell.
A. Crossley.	F. R. Humphreys.	E. T. Trevor.
A. H. Dodd.	E. G. Hunt.	F. W. Turtle.
W. H. Dodd.	C. Metzgar.	T. Unicum.
H. C. Ensor.	E. W. Phillips.	

Royal College of Surgeons.

Final Examination for the Fellowship.

W. A. Lane, M.S. | J. Poland.

First Examination for the Fellowship.

A. E. Larking.	F. N. Pedley.	F. Heatherley.
C. D. Muspratt.	A. E. Taylor.	J. T. J. Morrison.
C. Gross.	H. G. Dixon.	R. A. Baillie.
W. Fowler.	T. H. Miller.	J. J. D. Vernon.
W. L. Blight.		

Final Examination for the Membership.

January.

J. A. P. Price.	P. Pigott.	G. L. L. Lawson.
Q. R. Veitch.	F. C. Payne.	E. H. Booth.
C. T. Griffiths.	Geo. Utting.	H. A. Fotherby.
E. T. Trevor.	L. E. W. Stephens.	R. H. S. Spicer.
A. G. Wildey.		

April.

L. W. S. Beales.	C. Everest.	J. B. Trapp.
H. E. Rowell.	H. P. Berry, M.B.	H. A. Phillips.
L. A. Dunn.	J. H. Booth.	J. W. Hodgson.
W. C. Hearnden.	F. E. Hubbard.	W. D. J. Morris.
R. Beswick.	J. C. Pincott.	

July.

A. T. Perkins.	W. T. F. Davies, M.B.	H. C. Dixon.
W. E. Audland.	F. Eastes.	J. B. Berry.
E. S. Tresidder.	J. H. Gibson.	W. T. Harris.
J. B. Howell.	W. Spong.	G. S. Pollard.
T. Carr.		

November.

J. H. Lister.	J. A. Fraser.	H. R. Mead.
J. O. Littlewood.	T. Cardwell.	A. C. E. Woodhouse.
W. Hind, M.B.	J. Harrison.	A. W. C. Peskett.
H. T. Sells.	G. H. Kinch.	J. C. Underwood.
R. Cuff, M.B.	E. E. Masters.	S. Worthington, M.B.
W. R. Dakin, M.D.		

First Examination for the Membership.

January.

W. A. Shelswell.	J. W. Harris.	G. P. Wornum.
G. E. Palmer.	A. E. Price.	G. H. H. Fuller.
H. St. G. S. Hore.	L. F. Childe.	F. R. B. Bishopp.
G. De'Ath.	R. A. Bindley.	A. A. Jeyes.
T. B. Jacobson.		

April and May.

G. E. C. Anderson.	S. E. Prall.	E. P. Mourilyan.
W. E. P. Phillips.	J. H. Sellick.	J. Emery.
E. W. Phillips.	S. H. Agar.	F. W. Mawby.
A. H. Tubby.	J. H. Blight.	W. L. Rhys.
W. H. Bowes.	C. F. Wakefield.	S. R. Alexander.
C. Caldecott.	C. P. Walker.	R. Moody-Ward.
J. Chadwick.	W. H. Dodd.	G. T. Cattell.

466 *Gentlemen admitted to Practice, &c., in the year 1882.*

G. B. Harrop.	F. W. Welstead.	W. L. Braddon.
R. M. H. Randell.	E. K. Alderson.	T. N. Swindlehurst.
J. G. Harsant.	T. John.	M. M. Hailey.
F. W. Langridge.	W. J. Lee.	R. Carver.
R. J. Kerby.	F. R. Humphreys.	

July.

E. W. Goodall.	T. R. Rolston.	J. A. Fox.
J. D. Hughes.	H. M. Addison.	E. O. Greenwood.
J. D. Howe.	W. Lansdale.	G. C. Stamper.
E. S. Marder.	A. H. Fowler.	E. O. Hare.
M. W. Oldham.	J. Sandoe.	H. G. Hilbers.
H. J. Roberts.	W. F. Tronson.	F. H. Knaggs.
H. Vallance.	E. R. B. Archer.	G. R. M. Pollard.
A. W. Webb.	C. A. McAnally.	P. O. O. Billups.
R. Kœttlitz.	C. Fryer.	

November.

R. Denman.	R. G. Silverlock.	E. Goodall.
------------	-------------------	-------------

Apothecaries' Society.

Final Examination for the Licence.

W. E. Fielden, M.D.	H. W. Hart.	T. B. Winter.
L. E. W. Stephens.	J. O. Littlewood.	J. H. H. Manley.
E. H. Booth, M.B.	G. H. Graham.	A. G. Wildey.
L. A. Dunn, M.B.,	R. P. Samut.	A. W. C. Peskett.
B.S.	W. Watson.	J. H. Champ, M.B.
E. T. Trevor.	J. H. Lister.	C. Harrison.
D. T. Key.	R. F. Cox.	A. L. Tireman.
T. R. Atkinson.	S. Worthington, M.B.	A. J. Dalton.
J. W. Nicholson.	E. J. Muddle.	M. O'Kane, M.B.
M. J. Hart.	H. G. Plimmer.	W. S. N. Shorthouse.
J. B. Berry.	B. P. Bartlett.	

First Examination for the Licence.

F. V. Duckworth.	J. G. Milnes.	A. G. Wildey.
F. E. Hubbard.	T. Cardwell.	W. H. Carrington.
W. J. C. Tomalin.	A. J. Carter.	H. G. Plimmer.
W. Watson.	C. S. Jago.	G. W. B. Slader.
J. O. Littlewood.	E. J. Muddle.	E. W. Phillips.

University of London.

Matriculation Examination.

January.

H. L. Hart.	H. V. Hickman.
S. F. Holloway.	A. Scott.

June.

J. H. Barber.	T. Wilson-Smith.
---------------	------------------

MEDALLISTS AND PRIZEMEN, 1881-82.

JULY, 1882.

The Treasurer's Gold Medal for Medicine.

Lauriston Elgie Shaw, St. Leonards-on-Sea.

The Treasurer's Gold Medal for Surgery.

John Alfred Parry Price, Brecon.

Beanev Prize for Pathology.

John Alfred Parry Price, Brecon.

Michael Harris Prize for Anatomy.

Geo. Elliott Caldwell Anderson, Oudtsboorn, Cape Colony.

Fourth Year's Students.

Louis Albert Dunn, Brighton, First Prize, £25.

Thomas Carr, Brixton, Second Prize, £10.

Wheelton Hind, Honington, Certificate.

Third Year's Students.

Edgar Ernest Masters, Lewisham, First Prize, £25.

Francis Barclay Willmer Phillips, Brighton, Second Prize, £10.

William Watson, Rochester, Certificate.

Albert Martin, Wellington, New Zealand, Certificate.

George Arthur Johnson, Watlington, Oxon, Certificate.

Second Year's Students.

Joseph Hoare Prizes.

George Elliott Caldwell Anderson, Oudtsboorn, Cape Colony,
First Prize, £25.

Reginald Maurice H. Randell, Sydenham, Second Prize, £10.

Alfred Herbert Tubby, Kennington, Certificate.

Christopher Frank Wakefield, Camberwell, Certificate.

William Henry Bowes, Herne Bay, Certificate.

James Chadwick, York, Certificate.

Charles Caldecott, Basingstoke, Certificate.

Ernest Willmer Phillips, Brighton, Certificate.

James Henderson Sellick, Reigate, Certificate.

First Year's Students.

John Wychenford Washbourn, Gloucester, First Prize, £50.

Sydney Wachter, Canterbury, Second Prize, £25.

Frederick Lever, Epsom, Certificate.

Frederick William Winckworth, Bath, Certificate.

Samuel Walshe Owen, Peckham, Certificate.

Alfred Edward Price, Pontypridd, Certificate.

SEPTEMBER, 1882.

Open Scholarship in Arts.

John Lloyd Roberts.

Open Scholarship in Science.

William Frederick Clarke.

Medical Appointments held in the year 1882.

Durham Medical Society.

Session, 1882-83.

Honorary President—Dr. WILKS.

Presidents.

1881-82. M.D. E. H. Booth, M.B., H. Blatherwick, M.B., Thomas Carr, H. C. E. Cooper, B.S., J. W. Hodgson, B. A. Milligan, M.B., G. N. Pitt, M.A., M.D., J. A. P. Price, M.B., W. W. Prynn, B. N. Rake, M.D., L. E. W. Stephens, and J. B. Trapp.

1882-83. C. J. Symonds, M.S.; R. E. Carrington, M.D.

FELLOWS FOR THE SESSION, 1881-82.

1881-82. E. H. Booth, M.B., for his Paper on "Rickets."
H. C. E. Cooper, B.S., for his Paper on "The Role of Pigment in the Skin."
G. N. Pitt, M.B., for his Essay on the "Functions of the Sympathetic Nerve."
J. A. P. Price, M.B., as the Member who distinguished himself in the Debates of the Session.

CLINICAL APPOINTMENTS HELD IN THE YEAR 1882.

RESIDENT HOUSE PHYSICIANS.

E. A. Stirling	H. G. Bassett, M.B.
W. H. C. Newman, M.B.	J. I. Boswell.
H. H. Arnold, M.B.	T. M. Day.

RESIDENT HOUSE SURGEONS.

W. W. Pitt	L. Burroughs.
H. G. Adcock	R. A. Milligan.
R. N. Rake, M.D.	J. B. Trapp.

RESIDENT OBSTETRIC ASSISTANTS.

H. A. Clowes	E. H. Booth, M.B.	W. C. Dendy.
O. J. Currie, M.B.	L. Stokes.	H. Blatherwick.
J. I. Boswell	J. W. Hodgson.	G. N. Pitt, M.D.
E. L. Adeney, M.D.	J. A. P. Price, M.B.	L. E. Shaw, M.D.

SURGEONS' DRESSERS.

W. R. Dakin, M.D.	E. T. Trevor.	H. C. Dixon.
J. A. Fraser.	J. B. Howell.	L. A. Dunn, M.B., B.S.
W. H. Hart.	G. N. Pitt, M.D.	R. J. Ryle.
H. T. Sells.	G. Longman.	W. Hind, M.B., B.S.
T. Carr.	H. P. Berry, M.B.	W. T. F. Davies, M.B.
J. Dowson.	J. I. Palmer.	S. Worthington, M.B.
W. Growse.		

CLINICAL ASSISTANTS.

H. Blatherwick.	J. H. Lister.	H. P. Berry, M.B.
R. H. Perks.	J. A. P. Price, M.B.	W. Growse.
S. O. Stuart.	J. E. Viney.	E. T. Trevor.
W. C. Dendy.	W. D. J. Morris.	E. Roberts.
E. L. Adeney, M.D.	J. M. Prendergast,	E. S. Tresidder.
E. H. Booth, M.B.	M.D.	T. Carr.
J. H. Nicholson.		

DRESSERS IN THE EYE WARDS.

J. I. Boswell.	W. H. W. Strachan.	J. J. Udale.
H. E. Richardson.	H. C. Dixon.	E. H. Booth, M.B.
J. M. Owen.	J. B. Trapp.	H. T. Sells.
W. Hodgson.	R. H. Perks.	L. Stokes.
L. E. Shaw, M.D.	W. C. Dendy.	J. A. Fraser.
G. S. Pollard.	H. Blatherwick.	F. Eastes.

DENTAL SURGEONS' DRESSERS.

D. T. Edmunds.	R. J. Ryle.	H. W. Whyte.
S. Mundell.	H. E. M. Long.	M. A. Muirhead.
R. W. Brogden.	C. E. Bean.	J. H. Sellick.
H. C. Ensor.	A. A. Jeyes.	

MEDICAL WARD CLERKS.

W. D. Smallpeice.	H. P. Keatinge.	A. P. H. Griffiths.
H. W. Pigeon.	J. H. Targett.	A. L. Scott.
H. A. B. Davies.	W. Hind.	J. C. Bates.
E. D. Minter.	R. Cuff.	St. J. O. Rands.
S. Worthington.	T. B. Winter.	A. L. Paliologus.
H. H. Wright.	C. H. L. Meyer.	C. Y. Shuter.
J. B. Berry.	W. T. F. Davies.	W. H. Moore.
A. E. C. Woodhouse.	G. Kendall.	W. L. W. Marshall.
J. P. Martin.	E. R. S. Lipscomb.	S. B. A. Edsall.
H. R. Mead.	R. Browne.	A. T. F. Brown.
L. A. Dunn.	A. L. Tireman.	J. H. Cox.
J. J. Prendergast.	M. Parry-Jones.	M. A. Muirhead.
M. O'Kane.	G. H. Graham.	A. J. Dalton.
E. S. Tresidder.	R. L. Knaggs.	H. C. Ensor.
E. Roberts.	E. O. Newland.	T. B. Jacobson.
H. Howard.	D. T. Lewis.	J. V. Salvage.
T. F. B. Palmer.	A. J. Carter.	E. Linnell.
L. Powell.	L. J. Kidd.	A. S. Topham.
H. W. Moor.	W. W. Floyer.	F. B. W. Phillips.
J. A. Marsden.	G. W. B. Slader.	J. H. H. Manley.
E. J. Wenyon.	S. R. Thomas.	W. Watson.
J. H. H. Williams.	F. Pearse.	

ASSISTANT SURGEONS' DRESSERS.

E. Wakelam.	A. J. Carter.	M. Parry-Jones.
A. L. Tireman.	A. W. Clark.	W. D. Smallpeice.
A. L. Scott.	S. R. Thomas.	R. W. Brogden.
H. C. Ensor.	L. J. Kidd.	J. H. Targett.
M. A. Muirhead.	G. A. Johnson.	W. A. Aikin.
J. H. H. Williams.	R. A. Baillie.	M. Carnelley.
F. B. W. Phillips.	E. Linnell.	C. Titley.
T. B. Winter.	O. Y. Shuter.	W. H. Brenton.
F. Pearse.	D. T. Lewis.	A. C. Deare.
A. T. F. Brown.	St. J. O. Rands.	G. F. Hugill.
E. G. Hunt.	S. B. A. Edsall.	T. B. Jacobson.
R. Cuff.	W. Watson.	E. S. Jago.
A. S. Topham.	J. W. Sanders.	H. Howard.
A. Martin.	E. E. Masters.	A. E. Larking.
R. L. Knaggs.	A. G. Minns.	G. C. Stamper.
W. L. W. Marshall.	C. H. L. Meyer.	W. T. Hodge.
W. Hind.	A. H. Dodd.	E. W. Simmons.
H. P. Keatinge.	H. I. Tresidder.	W. A. Shelswell.
W. T. F. Davies.	T. H. Miller.	E. J. Wenyon.
J. A. Marsden.	E. Sharpley.	A. M. Sutton.
W. W. Floyer.	H. E. Counsell.	H. J. Jones.
J. C. Bates.	A. Linnell.	A. A. Jeyes.
E. D. Minter.	H. W. Whyte.	R. W. Murray.
G. W. B. Slader.	A. Martin.	W. Fowler.
J. P. Martin.	J. H. H. Manley.	J. W. Harris.
C. E. Bean.	J. J. Parsons.	T. N. Swindlehurst.
A. L. Paliologus.	J. V. Salvage.	

DRESSERS IN THE SURGERY.

H. W. Whyte.	A. Martin.	G. E. Stewart.
R. W. Brogden.	J. Harvey.	H. Lamb.
S. R. Thomas.	E. Sharpley.	R. W. Murray.
W. W. Floyer.	W. T. Hodge.	H. Howard.
A. E. Taylor.	A. Linnell.	C. F. Wakefield.
T. H. Miller.	F. B. W. Phillips.	F. W. Welstead.
W. A. Aikin.	H. I. Tresidder.	R. J. Cook.
A. G. Minns.	J. J. D. Vernon.	G. R. M. Pollard.
W. H. Brenton.	R. T. Westbrook.	L. F. Childe.
C. E. Bean.	H. T. Jones.	J. Emery.
J. H. H. Manley.	H. E. Counsell.	F. R. Humphreys.
W. Fowler.	G. E. Palmer.	M. M. Hailey.
A. H. Dodd.	A. A. Jeyes.	W. H. Dodd.
A. W. Clark.	E. J. Wenyon.	F. W. Mawby.
C. D. Muspratt.	J. H. Targett.	W. L. Blight.
J. I. Parsons.	A. E. Price.	W. I. Watson.
A. C. Deare.	H. St. G. S. Hore.	C. P. Walker.
H. J. Jones.	J. W. Harris.	R. L. Knaggs.
T. N. Swindlehurst.	E. W. Simmons.	R. J. Kerby.
T. B. Jacobson.	G. H. De'Ath.	A. H. Tubby.
M. Carnelley.	G. P. Wornum.	E. W. Phillips.
E. H. Armitage.	G. H. Rodman.	G. T. Cattell.
C. Titley.	W. A. Shelswell.	F. G. F. Chittenden.
E. S. Jago.	S. H. Agar.	W. L. Braddon.
G. F. Hugill.		

AURAL SURGEON'S DRESSERS.

C. Y. Shuter.	H. C. Ensor.	A. H. Dodd.
W. Watson.	R. J. Ryle.	G. H. Graham.
R. W. Brogden.	J. H. Gibson.	J. H. Cox.
L. McEwan Anderson.	J. O. Littlewood.	H. H. Wright.

ASSISTANT PHYSICIANS' CLERKS.

W. J. C. Tomalin.	J. J. Prendergast.	G. Kendall.
R. P. Samut.	W. D. Smallpiece.	G. H. Graham.
D. T. Key.	E. Roberts.	W. R. Etches.
G. H. Kinch.	M. O'Kane.	E. S. Tresidder.
Q. R. Veitch.	H. R. Mead.	C. Y. Shuter.
H. P. Berry.	W. E. Rudd.	L. McE. Anderson.
C. S. Harper.	T. B. Winter.	W. L. W. Marshall.
E. Apthorp.	R. H. Browne.	A. P. H. Griffiths.
W. E. Audland.	W. E. Fielden.	A. J. Carter.
J. J. Udale.	E. L. Adeney.	J. A. Marsden.
W. Wilson.	E. R. S. Lipscomb.	A. L. Tireman.

POST-MORTEM CLERKS.

J. H. Champ.	H. G. Plimmer.	G. Kendall.
S. B. A. Edsall.	A. J. Dalton.	L. J. Kidd.
W. E. Audland.	J. I. Palmer.	S. E. Prall.
W. R. Etches.	H. H. Wright.	W. L. W. Marshall.
L. McE. Anderson.	E. Wakelam.	G. W. B. Slader.
J. M. Prendergast.	J. P. Martin.	C. A. McAnally.
J. H. Gibson.	G. A. Johnson.	C. Y. Shuter.
S. Worthington.	W. Wilson.	

OBSTETRIC OUT-PATIENTS CLERKS.

H. L. Cortis.	E. Wakelam.	A. W. Clark.
C. E. Beebe.	W. R. Etches.	W. T. Hodge.
W. Wilson.	M. A. Muirhead.	T. H. Miller.
W. T. Harris.	E. S. Tresidder.	J. B. Howell.
W. J. C. Tomalin.	H. Gard.	R. P. Samut.
W. Watson.	M. Carnelley.	H. E. Counsell.
D. T. Key.	F. B. W. Phillips.	H. H. Wright.
W. Fowler.	T. Carr.	J. P. Martin.

OBSTETRIC WARD CLERKS.

J. B. Howell.	E. E. Masters.	E. Linnell.
G. H. Graham.	J. H. H. Manley.	St. J. O. Rands.
E. T. Trevor.	W. Fowler.	A. L. Scott.
A. T. F. Brown.	A. S. Topham.	H. C. E. Cooper.

EXTERN OBSTETRIC ATTENDANTS.

A. P. H. Griffiths.	G. W. B. Slader.	A. L. Scott.
G. A. Johnson.	H. R. Mead.	F. B. W. Phillips.
E. E. Masters.	J. J. Prendergast.	A. G. Minns.
A. J. Dalton.	E. S. Tresidder.	J. I. Parsons.
C. E. Beebe.	E. Roberts.	A. T. F. Brown.
R. T. Westbrook.	E. G. Hunt.	A. H. Dodd.
G. H. De'Ath.	M. O'Kane.	W. H. Brenton.
St. J. O. Rands.	J. H. H. Manley.	A. C. Deare.
J. W. Harris.	H. I. Tresidder.	W. Hind.

INTERNAL MEDICINE ATTENDANTS (continued).

W. F. Gier	S. H. Agar.	W. H. Bowes.
E. C. H.	C. H. L. Meyer.	H. C. E. Cooper.
R. L. Rogers	C. P. Walker.	L. Powell.
T. B. White.	W. J. Lee.	A. Green.
A. W. Clark	A. L. Palibogua.	E. H. Armitage.
A. L. Thomas	R. Moody-Ward.	C. S. Spong.
W. T. Hodge	H. R. Counsell.	L. J. Kidd.
C. E. Smith	H. Lamb.	F. W. H. Penfold.
J. Harvey	C. D. Muspratt.	H. J. Jones.
J. H. Targett	G. E. Palmer.	J. V. Salvage.
E. S. Jago.	J. C. Bates.	

SURGICAL WARD CLERKS.

H. Gard.	R. W. Murray.	F. W. Welstead.
D. T. Lewis	G. P. Wornum.	F. W. Mawby.
H. H. W. Burton.	H. Lamb.	W. E. P. Phillips.
L. J. Kidd.	G. E. Stewart.	J. Emery.
A. J. Carter.	G. H. Bodman.	H. H. Du Boulay.
E. J. Weyman.	J. W. Harris.	H. G. Hilbera.
R. A. Bindley.	W. A. Shelswell.	F. W. H. Penfold.
M. Carnelley.	H. St. G. S. Hore.	E. R. B. Archer.
J. H. Targett.	A. E. Price.	C. S. Spong.
C. H. L. Meyer.	J. O. Downes.	E. C. Greenwood.
C. Tiley.	P. W. Turtle.	P. C. C. Billups.
E. Linnell.	T. N. Swindlehurst.	E. P. Mourilyan.
C. Y. Shuter.	H. J. Jones.	H. E. Jones.
M. Parry-Jones.	R. J. Cook.	J. A. Fox.
E. Sharpley.	H. J. Dring.	J. Harvey.
J. J. D. Vernon.	W. L. Blight.	S. H. Agar.
H. I. Tresidder.	W. H. Dodd.	R. A. Bindley.
W. I. Watson.	A. E. Larking.	R. Koettlitz.
E. S. Jago.	C. P. Walker.	M. W. Oldham.
E. H. Armitage.	W. M. Sheen.	H. J. Hillstead.
E. O. Newland.	W. L. Rhys.	A. H. Fowler.
G. E. Palmer.	J. Chadwick.	F. W. Langridge.
A. A. Jeyes.	A. H. Tubby.	R. M. H. Randell.
J. V. Salvage.	R. J. Kerby.	C. Fryer.
E. W. Simmons.	R. J. Cattell.	C. Caldecott.
H. Howard.	M. M. Hailey.	G. E. C. Anderson.
G. H. De'Ath.		

ASSISTANT SURGEONS' CLERKS.

E. S. Marder.	Y. M. J. Humphreys.	H. J. Roberts.
C. Caldecott.	J. F. Rymer.	F. H. Knaggs.
G. Rowell.	R. Capes.	A. E. Wilson.
F. W. Turtle.	E. Deane.	E. S. Blaker.
W. A. Shelswell.	A. E. Price.	G. A. Wybourn.
M. W. Oldham.	G. T. Cattell.	J. H. Blight.
W. F. Tronson.	F. G. F. Chittenden.	H. Stelfox.
J. W. Washbourn.	G. Todd.	H. W. Whyte.
C. G. Wallis.	E. B. Harris.	A. H. Dodd.
R. Creasy.	S. H. Seccombe.	G. E. C. Anderson.
W. M. Sheen.		

LIST
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1883.

University of Oxford.

First Examination for the Degree of Bachelor of Medicine.
A. W. Webb.

University of Cambridge.

Final Examination for the Degree of Bachelor of Medicine.

J. T. J. Morrison.		W. H. O. Newnham.
H. W. Pigeon.		G. N. Pitt.

Second Examination for the Degree of Bachelor of Medicine.

D. C. Trott.		F. Wyatt-Smith.
H. Lund.		F. Beard.

First Examination for the Degree of Bachelor of Medicine.

A. S. Taylor.

Final Examination for the Degree of Bachelor of Surgery.

J. T. J. Morrison.		R. L. Knaggs.
J. H. H. Manley.		W. Fowler.

University of London.

Examination for the Degree of Doctor of Medicine.

E. L. Adeney.

Obtained the Gold Medal.

W. R. Dakin.		L. E. Shaw.
W. E. Fielden.		R. S. Wainewright.

Final Examination for the Degree of Bachelor of Medicine.

First Division.

J. H. Champ.

*Obtained First-Class Honours in Medicine, First-Class Honours in Obstetric
Medicine, and First-Class Honours in Forensic Medicine.*

474 Gentlemen admitted to Degrees, &c., in the year 1883.

J. A. P. Price.

Obtained First-Class Honours in Forensic Medicine, and Honours in Medicine.

W. Hind.

Obtained Honours in Obstetric Medicine and in Forensic Medicine.

S. Worthington.

Obtained Honours in Medicine and in Forensic Medicine.

R. H. Booth.

L. A. Dunn.

|

A. Martin.

M. Parry-Jones.

Second Division.

C. H. L. Meyer.

Obtained Honours in Forensic Medicine.

J. H. Targett.

Obtained First-Class Honours in Forensic Medicine.

H. P. Berry.

R. Cuff.

|

W. T. F. Davies.

M. O'Kane.

E. J. Wenyon.

Intermediate Examination in Medicine.

First Division.

W. L. Braddon.

Obtained First-Class Honours in Materia Medica, and Honours in Organic Chemistry and in Physiology and Histology.

G. E. Halstead, B.Sc.

Obtained Honours in Anatomy and in Organic Chemistry.

F. Lever, B.Sc.

Obtained Honours in Materia Medica and in Physiology and Histology.

J. W. Washbourn.

Obtained the Exhibition and Gold Medal in Organic Chemistry, First-Class Honours in Anatomy, and Honours in Materia Medica.

A. E. Price.

Second Division.

L. F. Childe.

Obtained Honours in Materia Medica.

C. D. Muspratt.

Obtained Honours in Materia Medica.

W. Aikin.

E. W. Goodall.

J. G. Harsant.

|

F. Heatherley.

E. P. Mourilyan.

S. E. Prall.

A. M. Sutton.

Excluding Physiology.

E. Roberts.

Gentlemen admitted to Degrees, &c., in the year 1888. 475

Preliminary Scientific (M.B.) Examination.

First Division.

E. H. Starling.

Obtained the Exhibition in Inorganic Chemistry, and First-Class Honours in Botany and in Experimental Physics.

G. Black.

Obtained Honours in Inorganic Chemistry.

J. L. Roberts.

Obtained First-Class Honours in Inorganic Chemistry and Honours in Experimental Physics.

**H. J. Campbell.
H. E. Cuff.**

**R. A. Sawyer.
T. Wilson-Smith.**

Second Division.

**S. Bueno de Mesquita.
H. A. Edmonds.
E. Moss.
G. J. Padbury.
P. N. Randall.**

**F. P. Sarjant.
W. E. Tresidder.
A. H. Tubby.
H. E. Vincent.
F. S. Wood.**

Experimental Physics only.

S. F. Holloway.

Examination for the Degree of Master of Surgery.

W. A. Lane.

Final Examination for the Degree of Bachelor of Surgery.

First Division.

C. H. L. Meyer.

Obtained First-Class Honours in Surgery.

L. A. Dunn.

Obtained Honours in Surgery.

Second Division.

W. Hind.

Obtained Honours in Surgery.

University of Durham.

Final Examination for the Degree of Bachelor of Medicine.

**G. H. Rodman.
F. Eastes.
A. T. F. Brown.**

**H. P. Keatinge.
J. I. Parsons.
C. Y. Shuter.**

E. W. Simmons.

First Examination for the Degree of Bachelor of Medicine.

T. Carr.

271 *Examinations administered in Practice, &c., in the year 1883.*

University of Edinburgh.

First Examination for the Degree of Bachelor of Medicine.

H. J. Dinning.

B. Moorhouse.

Second Examination for the Degree of Bachelor of Medicine.

H. J. Dinning.

First Examination for the Degree of Bachelor of Medicine.

W. C. Spiller.

Scottish Medical Society.

Examination in February.

J. H. Pennington, 1974 marks.

T. Bicketts-Morse, 1974 marks.

Royal College of Physicians.

Examination for the Membership.

H. Lloyd M.

W. Hale White, M.D.

First Examination for the Licence.

H. H. Brown.

W. W. Lister.

A. Bolton.

V. A. Brown.

W. B. Arnold.

J. L. Boswell.

H. S. Brown.

T. W. L. Brown.

J. H. Cox.

H. E. Brown.

A. J. Brown.

J. A. Fraser.

H. E. Brown.

J. D. Brown.

R. L. Knaggs.

T. H. Brown.

J. C. Brown.

T. H. Miller.

H. A. Brown.

F. C. Payne.

M. F. Cock.

H. E. Brown.

H. I. Brown.

First Examination for the Licence.

February.

C. E. Brown.

H. St. G. S. Hore.

W. B. P. Phillips.

C. E. Brown.

E. Brown.

A. E. Price.

C. E. Brown.

W. L. W. Marshall.

W. L. Rhys.

C. E. Brown.

E. O. Newland.

W. S. N. Shorthouse.

H. C. Brown.

May.

M. A. Brown.

J. D. Hughes.

G. T. Rawnsley.

H. E. Brown.

W. J. Lee.

J. W. Sandoe.

J. H. Cox.

D. T. Lewis.

W. Spong.

J. A. Fraser.

M. W. O'Han.

August.

L. M. E. Anderson.

H. A. Forberby.

P. W. H. Penfold.

E. E. E. Anderson.

J. A. Fox.

H. V. Rake.

J. C. Fox.

P. O. W. Hailey.

A. L. Scott.

H. T. Fox.

C. S. Harper.

G. S. Shute.

P. C. C. Fox.

E. C. L. Hendriem.

C. R. Stedman.

W. L. Fox.

E. R. S. Lipscomb.

H. W. Whyte.

W. H. Fox.

J. W. F. Long.

W. W. Williams.

A. R. Garver.

C. A. Lumley.

S. A. Davies.

H. W. Drew.

J. A. Marsden.

J. Harvey.

October.

R. T. Bedford.

C. W. Hogarth.

J. F. Saunders.

F. C. Butt.

A. H. Johnston.

G. S. Schofield.

R. Capen.

P. W. Mawby.

A. Sutton.

M. G. Dundas.

J. R. J. Raywood.

Royal College of Surgeons.

Final Examination for the Fellowship.

C. Gross.		J. T. J. Morrison, M.B.
-----------	--	-------------------------

First Examination for the Fellowship.

W. L. Braddon.		R. M. H. Randell.
W. H. Bowes.		J. G. Harsant.

Final Examination for the Membership.

January.

E. G. Hunt.	M. A. Muirhead.	A. J. Dalton.
B. H. Lane.	J. H. Targett.	H. G. Plimmer.
J. A. Marsden.	J. W. F. Long.	M. O'Kane.
St. J. O. Rands.	H. W. Pigeon.	R. H. Browne.
H. P. Keatinge.	J. H. Champ.	

April.

G. A. Johnson.	W. Watson.	E. R. D. Fasken.
C. H. L. Meyer.	H. H. Wright.	J. F. Spong.
J. H. H. Williams.	W. R. Etches.	A. P. H. Griffiths.
W. E. Rudd.	J. W. Nicholson.	B. P. Bartlett.
L. McE. Anderson.	A. Martin.	T. B. Winter.
J. H. H. Manley.	W. S. N. Shorthouse.	E. Roberts.
R. J. Ryle.	W. D. Smallpeice.	

July.

G. H. Rodman.	A. T. F. Brown.	F. N. Shillingford.
C. Y. Shuter.	A. W. Clark.	A. S. Topham.
C. E. Beebe.	J. H. Cuolahan.	J. J. Faraker.
J. H. Cox.	W. W. Floyer.	A. L. Tireman.
A. Linnell.	G. Kendall.	W. H. Brenton.
F. B. W. Phillips.	E. Sharpley.	C. S. Jago.
H. I. Tresidder.	W. H. Moore.	W. J. Parkinson.

November.

H. A. B. Davies.	R. L. Knaggs.	J. P. Martin.
D. T. Edmunds.	J. V. Salvage.	E. W. Simmons.
G. H. Graham.	C. P. Walker.	A. E. Larking.
H. Moor.	F. A. A. Bush.	E. R. S. Lepscomb.
S. B. A. Edsall.	W. Fowler.	J. H. H. Williams.
W. Howard.		

First Examination for the Membership.

January.

P. O. W. Hailey.	A. Sutton.	W. H. Boger.
J. R. J. Raywood.	F. C. Butt.	E. C. Kingsford.
Y. M. J. Humphreys.	F. W. Foster.	G. G. O. Phillips.
J. A. Bradbury.	H. Lund.	G. Schofield.
P. Purnell.	J. A. Benson.	A. G. Mossop.
H. H. Du Boulay.	M. Yunge-Bateman.	T. Slater Jones.
F. E. Cave.	M. G. Dundas.	S. A. Mugford.
D. M. Evans.	L. Selway.	

478 *Gentlemen admitted to Practice, &c., in the year 1883.*

April and May.

J. W. Washbourn.	R. Creasy.	P. N. Randall.
E. W. Du Buisson.	J. Crisp.	C. Pollard.
H. K. Roper.	H. Gilford.	J. N. Phillips.
G. E. Halstead.	E. Deane.	H. Rockley.
A. E. Price.	F. Beard.	L. Bidwell.
F. Lever, B.Sc.	C. N. Graham.	F. R. Bolton.
S. Wachter.	R. J. Harvey.	G. S. Kendall.
G. Rowell.	J. S. Grose.	W. M. Sheen.
S. W. Owen.	C. G. Wallis.	B. C. Gowan.
G. F. Pollard.	E. W. Marshall.	A. E. Wilson.
C. Metzgar.	H. J. Blackler.	G. F. E. Morgan.
W. H. W. Elliot.	D. A. Waite.	

July.

E. S. Blaker.	A. Meyrick-Jones.	S. H. Seccombe.
R. Capes.	E. H. Lipscomb.	C. S. Simpson.
C. J. Cressy.	E. Lester.	A. Ward.
J. H. Collymore.	H. A. Reed.	W. P. West.
E. A. Farr.	G. H. Reynolds.	P. Meldrum.
E. B. Harris.		

November.

F. Pearse.	E. H. Brock.
R. T. Kent.	F. R. H. Potts.

Apothecaries' Society.

Final Examination for the Licence.

G. H. Rodman.	A. T. F. Brown.	E. Linnell.
A. P. H. Griffiths.	A. E. Taylor.	J. A. Marsden.
W. D. J. Morris.	J. H. Cox.	E. W. Simmons.
J. H. H. Williams.	C. E. Beebe.	J. W. F. Long.
F. E. Hubbard.	M. Parry-Jones.	E. R. S. Lipscomb.
G. W. B. Slader.	C. Y. Shuter.	S. H. Agar.
W. J. C. Tomalin.	T. H. Miller.	L. McE. Anderson.
W. C. Hearnden.	J. W. Harris.	C. Caldecott.
W. H. Brenton.	A. E. Larking.	C. E. Bean.
C. S. Jago.	A. H. Dodd.	

First Examination for the Licence.

W. C. Hearnden.	J. M. Griffin.
T. N. Swindlehurst.	J. W. F. Long.
W. P. West.	

University of London.

Matriculation Examination.

January.

C. S. Davis.	G. R. G. Malkin.
R. T. Temple.	A. H. Tubby.

June.

T. F. Ricketts.	G. F. Brown.
-----------------	--------------

MEDALLISTS AND PRIZEMEN, 1882-83.

JULY, 1883.

The Treasurer's Gold Medal for Medicine.

Thomas Carr, Brixton.

The Treasurer's Gold Medal for Surgery.

Wheelton Hind, Honington.

Gurney Hoare Prize for Clinical Study.

Wheelton Hind, Honington, } equal { £12 10s.
James Henry Targett, Salisbury, } equal { £12 10s.

Beaney Prize for Pathology.

Wheelton Hind, Honington.

Mackenzie Bacon Prize for Ophthalmoscopy.

Edward Roberts, Aberystwith.

Sands Cox Scholarship for Physiology.

Frederick Lever, Epsom.

Michael Harris Prize for Anatomy.

John Wychenford Washbourn, Gloucester.

Fourth Year's Students.

Albert Martin, Wellington, New Zealand, First Prize, £25.

George Hook Rodman, South Norwood, Second Prize, £10.

Herbert Edward Counsell, Camberwell, Certificate.

John Herbert Hawkins Manley, West Bromwich, Certificate.

Thomas Hugh Miller, Virginia, America, Certificate.

Walter Henry Dodd, Highbury, Certificate.

Arthur Ernest Larking, Tonbridge, Certificate.

Third Year's Students.

George Elliott Caldwell Anderson, Oudtsboorn, Cape Colony,
First Prize, £25.

Ernest Willmer Phillips, Brighton, Second Prize, £10.

William Henry Bowes, Herne Bay, Certificate.

Charles Caldecott, Basingstoke, Certificate.

Reginald Maurice Henry Randell, Sydenham, Certificate.

Second Year's Students.

Joseph Hoare Prizes.

John Wychenford Washbourn, Gloucester, First Prize, £25.

Sidney Wachter, Eddington, Canterbury, Second Prize, £10.

Frederick Lever, Epsom, Certificate.

Samuel Walshe Owen, Peckham, Certificate.

First Year's Students.

Henry William Drew, Southwark, First Prize, £50.

Frédéric François Burghard, Kensington, } equal { £12 10s.
Herbert Vaughan Rake, Fordingbridge, } equal { £12 10s.

Francis Stanhope Hawkins, Lamberhurst, Certificate.

Arnold Scott, Tulse Hill, Certificate.

William Frederick Clarke, Kensington, Certificate.

SEPTEMBER, 1883.

Open Scholarship in Arts.

George Herbert Pennell, Exeter.

Open Scholarship in Science.

Ernest Henry Starling, Bombay.

Pupils' Physical Society.

Session 1883-84.

Honorary President.—Dr. WILKS.

Presidents.

Messrs. G. E. C. Anderson, H. P. Berry, M.B., H. C. E. Cooper, W. Fowler, M.A., B.Sc., R. L. Knaggs, B.A., M.B., A. Martin, M.B., R. Moody-Ward, B.A., H. W. Pigeon, M.B., W. A. Slater, B.Sc., J. H. Targett, M.B., W. H. Bowes, R. J. Kerby, R. M. H. Randell, E. W. Phillips, J. H. Sellick, C. S. Spong, B.Sc., A. H. Tubby, J. W. Washbourn.

Honorary Secretaries.—R. E. CARRINGTON, M.D.; W. H. WHITE, M.D.

PRIZEMEN FOR THE SESSION, 1882-83.

To Mr. R. L. Knaggs, £10, for his Paper on "Cerebral Tumour."

To Mr. G. E. C. Anderson, £5, for his Essay on "The Histology and Physiology of the Blood."

To Mr. J. I. Parsons, £5, as the Member who distinguished himself most in the Debates of the Session.

CLINICAL APPOINTMENTS HELD IN THE YEAR 1883.

RESIDENT HOUSE PHYSICIANS.

O. J. Currie, M.B.	L. E. Shaw, M.D.
J. A. P. Price, M.B.	G. N. Pitt, M.B.
E. L. Adeney, M.D.	L. E. W. Stephens.

RESIDENT HOUSE SURGEONS.

H. Perks.	J. T. J. Morrison, M.B.
L. E. W. Stephens.	H. Blatherwick.
L. Stokes.	W. C. Dendy.

RESIDENT OBSTETRIC ASSISTANTS.

T. Carr.	L. E. W. Stephens.	H. H. Wright.
H. P. Berry, M.B.	W. Hind, M.B., B.S.	R. J. Ryle.
H. T. Sells.	L. A. Dunn, M.B., B.S.	H. W. Pigeon, M.B.
W. R. Dakin, M.D.	F. Eastes.	J. H. Targett, M.B.

SURGEONS' DRESSERS.

R. Cuff, M.B.	W. E. Audland.	A. Martin, M.B.
C. H. L. Meyer, M.B.	J. H. Targett, M.B.	M. A. Muirhead.
J. H. Champ, M.B.	G. Kendall.	G. H. Rodman, M.B.
J. O. Littlewood.	H. W. Pigeon, M.B.	C. Y. Shuter, M.B.
M. Parry-Jones, M.B.	R. L. Knaggs, M.B.	A. W. Clark.
M. O'Kane, M.B.	M. Carnelley.	R. W. Brogden.
J. V. Salvage.	H. P. Keatinge.	R. A. Baillie.
E. J. Wenyon, M.B.	F. B. W. Phillips.	J. H. H. Manley, M.B.

CLINICAL ASSISTANTS.

F. Eastes.	R. J. Ryle.	J. V. Salvage.
L. A. Dunn, M.B., B.S.	M. Parry-Jones, M.B.	M. Carnelley.
W. Hind, M.B., B.S.	R. Cuff, M.B.	W. E. Audland.
W. T. F. Davies, M.B.	C. H. L. Meyer, M.B.,	F. B. W. Phillips.
S. Worthington, M.B.	B.S.	G. Kendall.
H. G. Plimmer.	M. O'Kane, M.B.	A. Martin, M.B.
G. N. Pitt, M.D.	H. H. Wright.	

DRESSERS IN THE EYE WARDS.

J. C. Underwood.	G. A. Johnson.	R. P. Samut.
W. D. J. Morris.	H. C. Ensor.	H. W. Pigeon, M.B.
L. Burroughs.	W. L. W. Marshall.	A. C. Deare.
E. G. Hunt.	J. H. Champ, M.B.	S. Worthington, M.B.
E. Roberts.	J. A. P. Price, M.B.	W. T. F. Davies, M.B.
J. J. Prendergast.	J. H. Cox.	M. O'Kane, M.B.
J. B. Howell.	C. E. Bean.	C. H. L. Meyer, M.B.
H. H. Wright.	G. H. Graham.	B.S.

DENTAL SURGEONS' DRESSERS.

W. L. W. Marshall.	R. M. H. Randell.	J. F. Rymer.
E. D. Minter.	W. L. Blight	H. C. Ensor.
A. J. Carter.	H. St. G. S. Hore.	J. Harvey.
A. C. Deare.	J. J. D. Vernon.	W. H. Dodd.

MEDICAL WARD CLERKS.

G. E. Stewart.	W. T. Hodge.	C. S. Jago.
C. E. Bean.	T. H. Miller.	C. P. Walker.
E. E. Masters.	A. H. Dodd.	W. H. Dodd.
A. W. Clark.	R. W. Murray.	A. E. Larking.
G. A. Johnson.	J. H. Harris.	E. W. Phillips.
A. Linnell.	W. A. Aikin.	E. C. Greenwood.
H. I. Tresidder.	H. E. Counsell.	E. R. B. Archer.
A. C. Deare.	E. W. Simmons.	H. C. E. Cooper.
G. H. Rodman.	M. M. Hailey.	W. I. Watson.
J. I. Parsons.	E. H. Armitage.	A. H. Fowler.
W. H. Brenton.	H. St. G. S. Hore.	J. A. Fox.
G. F. Hugill.	G. E. Palmer.	W. L. Blight.
A. Martin.	A. E. Price.	F. R. Humphreys.
A. G. Minns.	T. A. Evans.	H. J. Hillstead.
E. Sharpley.	J. Harvey.	S. H. Agar.
G. P. Wornum.	H. G. Hilbers.	A. E. Taylor.
T. N. Swindlehurst.	H. W. Whyte.	R. J. Kerby.
M. Carnelley.	C. S. Spong, B.Sc.	R. J. Cook.
R. A. Baillie.	F. W. H. Penfold.	G. E. C. Anderson.
A. A. Jeyes.	G. H. De'Ath.	C. Caldecott.
G. C. Stamper.	W. A. Shelswell.	R. M. H. Randell.
R. W. Brogden.	H. Lamb.	W. M. Sheen.
H. Howard.	J. J. D. Vernon.	E. S. Marder.
W. Fowler.	R. Koettlitz.	C. D. Muspratt.

ASSISTANT SURGEONS' DRESSERS.

W. E. Wilson.	G. E. Stewart.	M. W. Oldham.
F. W. Mawby.	G. R. M. P. Card.	W. M. Sheen.
W. E. Jones.	G. P. Warran.	H. H. Du Boulay.
T. F. S. Palmer.	G. H. Bolman.	J. D. Howe.
J. A. Turner.	G. E. C. Anderson.	R. Denman.
R. J. Cook.	A. E. Taylor.	F. W. Mawby.
H. E. Jones.	H. C. E. Cooper.	C. Fryer.
W. E. Smith.	E. C. Greenwood.	W. H. Bowes.
A. E. Jones.	H. J. Hindstead.	R. A. Bindley.
H. E. Alexander.	C. Calhoun.	W. J. Lee.
H. E. Palmer.	R. J. Kerby.	G. G. O. Phillips.
H. E. W. Jones.	S. H. Agar.	R. G. Silverlock.
J. E. Taylor.	F. R. Humphreys.	F. C. Butt.
R. A. Palmer.	A. H. Fowler.	F. W. H. Penfold.
H. E. Mawby.	L. F. Child.	F. W. Langridge.
H. E. Jones.	E. P. McCarthy.	W. E. P. Phillips.
H. E. Jones.	P. M. Earle.	A. H. Tubby.
M. M. Taylor.	C. T. Carrall.	E. W. Phillips.
H. E. S. S. Jones.	W. L. Rhys.	F. R. B. Bisschopp.
H. E. Jones.	W. L. Braddon.	H. G. Dixon.
J. A. Fox.	E. S. Marder.	J. A. Bradbury.
R. T. Mawby.	R. Moody-Ward.	F. W. Foster.
H. E. S. Jones.	F. H. Knagga.	F. E. Cave.
P. C. C. Jones.	J. H. Selick.	F. R. Bolton.

DRESSERS IN THE SURGERY.

E. R. B. Archer.	F. H. Knagga.	W. F. Tronson.
E. C. Greenwood.	J. H. Bight.	J. Sandoe.
H. C. E. Cooper.	E. S. Marder.	W. H. Boger.
P. C. C. Phillips.	W. J. Lee.	A. G. Mossop.
E. P. McCarthy.	W. L. Rhys.	H. G. Dixon.
A. H. Fowler.	J. H. Selick.	H. Lund.
R. A. Palmer.	W. L. Braddon.	F. R. B. Bisschopp.
H. J. Hindstead.	E. Goodall.	G. Schofield.
J. A. Fox.	W. H. Bowes.	A. Sutton.
S. R. Alexander.	E. W. Goodall.	M. G. Dundas.
F. W. Langridge.	J. D. Howe.	G. F. Pollard.
R. M. H. Randall.	J. Vallance.	P. O. W. Hailey.
G. E. C. Anderson.	F. R. Bolton.	J. Crisp.
C. Fryer.	R. G. Silverlock.	H. J. Roberts.
C. Calhoun.	S. A. Mugford.	R. Creasy.
W. E. P. Phillips.	J. A. Bradbury.	E. S. Blaker.
A. W. Webb.	G. G. O. Phillips.	W. P. West.
S. E. Prall.	F. W. Foster.	E. Deane.
P. M. Earle.	J. R. J. Raywood.	A. Meyrick-Jones.
A. R. Carter.	Y. M. J. Humphreys.	S. Wachter.
F. Heatherley.	E. K. Alderson.	H. Gilford.
A. Green.	H. M. Addison.	J. D. Hughes.
R. Denman.	H. T. Benson.	G. B. Harrop.
M. W. Oldham.	L. Selway.	E. H. Lipscomb.
H. H. Du Boulay.	F. C. Butt.	W. S. Brown.
T. Slater Jones.		

AURAL SURGEON'S DRESSERS.

A. L. Tireman.	E. Roberts.	H. P. Berry.
A. M. Sutton.	J. J. Prendergast.	W. Wilson.
J. P. Martin.	J. V. Salvage.	R. L. Knaggs.
C. D. Muspratt.	H. E. Counsell.	H. P. Keatinge.

ASSISTANT PHYSICIANS' CLERKS.

A. L. Scott.	H. I. Tresidder.	H. E. Counsell.
R. J. Kerby.	G. F. Hugill.	J. I. Parsons.
T. B. Winter.	W. H. Brenton.	W. Fowler.
St. J. O. Rands.	T. N. Swindlehurst.	J. P. Martin.
J. P. Martin.	A. L. Paliologus.	G. T. Cattell.
S. B. A. Edsall.	J. W. Harris.	W. A. Shelswell.
F. Pearse.	W. H. Dodd.	M. Parry-Jones.
M. A. Muirhead.	C. S. Spong, B.Sc.	W. L. Blight.
J. H. H. Williams.	F. W. H. Penfold.	A. H. Fowler.
C. Fryer.	A. H. Dodd.	H. W. Whyte.
A. J. Dalton.	E. H. Armitage.	A. A. Jeyes.
R. A. Baillie.	W. T. F. Davies.	S. A. Mugford.

POST-MORTEM CLERKS.

J. C. Bates.	W. D. Smallpeice.	C. S. Spong, B.Sc.
J. H. H. Manley.	J. H. Cuolahan.	G. H. De'Ath.
F. W. H. Penfold.	H. Gilford.	E. A. Farr.
F. B. W. Phillips.	T. N. Swindlehurst.	H. Vallance.
W. Growse.	A. W. Webb.	H. E. Counsell.
E. Linnell.	J. H. Targett.	E. Sharpley.
H. E. Jones.		

OBSTETRIC OUT-PATIENTS CLERKS.

W. D. Smallpeice.	W. W. Floyer.	J. W. Harris.
R. H. Browne.	J. H. H. Manley.	H. Vallance.
R. L. Knaggs.	G. F. Cattell.	F. Heatherley.
W. A. Shelswell.	C. Y. Shuter.	G. E. Stewart.
A. E. Larking.	R. W. Murray.	C. P. Walker.
E. S. Jago.	A. H. Dodd.	T. John.
C. F. Wakefield.	T. Slater Jones.	C. E. Bean.
H. T. Benson.	A. J. Dalton.	Y. M. J. Humphreys.

OBSTETRIC WARD CLERKS.

C. S. Spong, B.Sc.	W. I. Watson.	W. T. Hodge.
H. C. Ensor.	G. A. Johnson.	J. R. J. Raywood.
S. H. Agar.	W. W. Floyer.	W. H. Dodd.
J. I. Parsons.	J. C. Bates.	

EXTERN OBSTETRIC ATTENDANTS.

R. J. Kerby.	M. W. Oldham.	G. C. Stamper.
A. Carver.	W. A. Shelswell.	A. Martin.
D. T. Lewis.	A. E. Larking.	W. E. P. Phillips.
C. Caldecott.	G. T. Cattell.	M. Dundas.
J. A. Fox.	E. Sharpley.	G. Schofield.
G. G. O. Phillips.	A. Linnell.	W. L. Blight.

EXTERNAL OBSTETRIC ATTENDANTS (continued).

R. A. Baillie.	J. D. Hughes.	E. W. Du Buisson.
G. F. Hugill.	G. Todd.	W. H. Boger.
W. I. Watson.	F. E. Cave.	L. Selway.
P. W. Mawby.	J. H. Collymore.	F. Heatherley.
R. J. Cook.	F. W. Foster.	A. E. Price.
A. Sutton.	S. A. Mugford.	G. F. E. Morgan.
W. Lansdale.	G. Pender-Smith.	E. R. B. Archer.
A. S. Kerby.	F. H. Knagga.	G. E. Halstead.
E. J. Johnstone.	A. H. Fowler.	J. J. D. Vernon.
H. St. G. S. Hore.	E. A. Farr.	G. E. C. Anderson.
M. M. Hailey.	W. F. Tronson.	C. Pink.
R. W. Baggien.	J. D. Howe.	H. J. Hillstead.
G. R. M. Pollard.	F. R. Humphreys.	T. F. Christal.
P. A. McCarthy.	H. J. Blackler.	A. E. Taylor.

SURGICAL WARD CLERKS.

G. R. M. Pollard.	H. G. Dixon.	E. H. Lipscomb.
P. M. Earle.	A. G. Mossop.	G. Schofield.
A. Green.	W. F. Tronson.	C. E. Player.
L. F. Child.	F. C. Butt.	W. S. Brown.
R. Denman.	W. H. Boger.	W. Fowler.
F. R. Humphreys.	Y. M. J. Humphreys.	J. H. Collymore.
F. H. Knagga.	L. Selway.	C. J. Cressy.
A. E. Taylor.	J. R. J. Raywood.	E. Lester.
F. Heatherley.	H. M. Addison.	W. A. Slater.
H. Vallance.	H. Lund.	E. Goodall.
E. S. Marder.	E. K. Alderson.	J. S. Grose.
W. J. Lee.	J. W. Sandoe.	T. Slater Jones.
J. H. Selick.	R. Moody Ward.	J. G. Harsant.
W. L. Bradion.	T. R. B. Bishopp.	H. A. Reed.
J. D. Hughes.	C. N. Graham.	E. W. Goodall.
T. R. Rolston.	M. G. Dundas.	A. W. Webb.
J. D. Howe.	A. Sutton.	S. E. Prall.
E. W. Phillips.	J. H. Blight.	S. B. Alexander.
E. C. Hare.	G. B. Harrop.	C. G. Wallis.
W. H. Bowes.	G. F. Pollard.	B. C. Gowan.
F. E. Cave.	W. Lansdale.	P. N. Randall.
D. M. Evans.	P. O. W. Hailey.	H. K. Roper.
S. A. Mugford.	W. H. W. Elliot.	G. Rowell.
J. A. Bradbury.	E. W. Du Buisson.	F. Beard.
F. W. Foster.	J. Crisp.	E. C. Kingsford.
R. G. Silverlock.	S. W. Owen.	H. T. Benson.
G. G. O. Phillips.	R. Creasy.	

ASSISTANT SURGEONS' CLERKS.

S. F. Holloway.	H. Gilford.	F. W. Farr.
J. C. Bell.	P. Paget.	P. McCarthy.
E. W. Goodall.	S. B. Alexander.	P. M. Earle.
T. Slater Jones.	H. J. Campbell.	H. J. Hillstead.
J. Crisp.	A. H. Johnston.	G. Niven.
G. H. Metcalfe.	F. E. Cave.	E. L. Heidepriem.
H. N. Edwards.	H. E. Crook.	G. F. Hugill.
R. T. Wallace.	W. W. Floyer.	E. Moss.
W. Bett.	J. W. Harris.	A. Scott.
A. R. F. Everahed.	F. F. Burghard.	R. J. Kerby.

MEDALLISTS AND PRIZEMEN, 1883-84.

JULY, 1884.

The Treasurer's Gold Medal for Medicine.

Albert Martin, Wellington, New Zealand.

The Treasurer's Gold Medal for Surgery.

George Elliott Caldwell Anderson, Oudtsboorn, Cape Colony.

Gurney Hoare Prize for Clinical Medicine.

John Herbert Hawkins Manley, West Bromwich.

Beaney Prize for Pathology.

Albert Martin, Wellington, New Zealand.

Michael Harris Prize for Anatomy.

Herbert Vaughan Rake, Fordingbridge.

Francis Stanhope Hawkins, Lamberhurst, } *Proxime accesserunt.*
Arnold Scott, Tulse Hill, }

The Mackenzie Bacon Prize for Ophthalmoscopy.

James Henry Targett, Idmiston, Salisbury.

The Mackenzie Bacon Prize for Nervous Diseases.

William Leonard Braddon, Upton-on-Severn.

The Burdett Prize for Hygiene.

Hubert Housemayne du Boulay, Winchester.

Fourth Year's Students.

George Elliott Caldwell Anderson, Oudtsboorn, Cape Colony,
First Prize, £25.

William Leonard Braddon, Upton-on-Severn, Second Prize, £10.

William Henry Bowes, Herne Bay, Certificate.

Alfred Herbert Tubby, Kennington, Certificate.

Reginald Maurice H. Randell, Sydenham, Certificate.

Third Year's Students.

Frederick Lever, Epsom, First Prize, £25.

Sidney Wachter, Canterbury, Second Prize, £10.

Frederic Beard, Horton, Certificate.

Second Year's Students.

Henry William Drew, Southwark, First Prize, £25.

Frédéric François Burghard, Kensington, Second Prize, £10.

Arnold Scott, Tulse Hill, Certificate.

Cyrus Legg, Blackheath, Certificate.

First Year's Students.

Ernest Henry Starling, Bombay, First Prize, £50.

Guy Bellingham Smith, Lee, Second Prize, £25.

Herbert Edmund Cuff, Lewisham, Certificate.

Arlingham Carter, Gloucester, Certificate.

William Elliot Tresidder, West Dulwich, Certificate.

SEPTEMBER, 1884.

Open Scholarship in Arts.

James McDonald Gill, Lewisham.

Open Scholarship in Science.

Alfred Parkin, Hightown, Yorkshire.

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.

1884.

Consulting Physicians.

SIR WILLIAM FLEMING, M.D., F.R.C., F.R.S.; G. OWEN REES, M.D., F.R.S.

Physicians.

S. VANCE, M.D., F.R.S.; F. W. PATE, M.D., F.R.S.; W. MOXON, M.D.;
F. H. PEE-SHAY, M.D.

Assistant Physicians.

FREDERICK TAYLOR, M.D.; J. F. GOODHART, M.D.; F. A. MAHOMED, M.D.

Consulting Surgeons.

R. JACK, Esq.; J. BIRKETT, Esq.

Surgeons.

THOMAS BRYANT, Esq.; A. E. DUBRAN, Esq.; H. G. HOWER, M.S.;
N. DAVEN-COLLEY, M.C.

Assistant Surgeons.

A. CHARLES LUCAS, Esq.; J. H. GILLING-BIRD, M.B.; W. H. A. JACOBSON, Esq.;
C. J. STIMYER, M.S.

Consulting Obstetric Physicians.—HENRY OLDHAM, M.D.; J. BRAXTON
HICKS, M.D., F.R.S.

Obstetric Physician.—A. L. GALABIN, M.D.

Assistant Obstetric Physician.—P. HORROCKS, M.D.

Consulting Ophthalmic Surgeon.—C. RADER, Esq.

Ophthalmic Surgeon.—C. HIGGINS, Esq.

Assistant Ophthalmic Surgeon.—W. A. BRAILLY, M.D.

Dental Surgeon.—H. MOON, Esq.

Anal Surgeon.—W. LAIDLAW PEVER, Esq.

Medical Registrar.—R. E. CARRINGTON, M.D.

Surgical Registrar.—L. A. DUFF, M.B., B.S.

Instructor in Anæsthetics.—T. BIRD, M.A.

Curator of the Museum.—Dr. GOODHART.

Lying-in Charity.—Dr. A. L. GALABIN and Dr. HORROCKS.

Dean.—Dr. FREDERICK TAYLOR.

WINTER COURSES.

The Winter Session commences October 1st and ends March 31st.

LECTURES.

Medicine.—Dr. MOXON and Dr. PYE-SMITH.

Mondays, Wednesdays, and Fridays, at Three.

Clinical Medicine.—Dr. WILKS, Dr. PAVY, Dr. MOXON, and Dr. PYE-SMITH.

Saturdays, at Half-past One.

Surgery.—Mr. BRYANT and Mr. DURHAM.

Tuesdays and Thursdays, at Half-past Three, and Saturdays, at a Quarter to Three.

Clinical Surgery.—Mr. BRYANT, Mr. DURHAM, Mr. HOWSE, and Mr. DAVIES-COLLEY.

Wednesdays, at Half-past One.

Ophthalmic Surgery.—Mr. HIGGINS.

Wednesdays, at Three.

Anatomy, Descriptive and Surgical.—Mr. HOWSE and Mr. DAVIES-COLLEY.

Tuesdays, Wednesdays, Thursdays, and Fridays, at Nine.

Physiology and General Anatomy.—Mr. GOLDING-BIRD.

Mondays, Wednesdays, and Fridays, at a Quarter-past Four.

Clinical Lectures on Midwifery and Diseases of Women.—Dr. GALABIN.

Wednesdays, at Half-past One.

Chemistry.—Dr. DEBUS, F.R.S., and Dr. STEVENSON.

Tuesdays, Thursdays, and Saturdays, at Eleven.

Experimental Physics.—Prof. A. W. REINOLD, F.R.S.

Mondays and Wednesdays, at Eleven.

Comparative Anatomy and Zoology.—Dr. BRAILEY and Dr. PITT.

Mondays and Wednesdays, at a Quarter to Two.

DEMONSTRATIONS.

Practical Surgery.—Mr. LUCAS.

Surgical Classes.—Mr. JACOBSON, *Daily.*

Practical Obstetrics.—Dr. HORROCKS.

Practical Anatomy.—Dr. W. H. WHITE, Mr. W. A. LANE, and Mr. J. POLAND, *Demonstrators.*

And two Assistant Demonstrators.

Morbid Anatomy.—Dr. GOODHART and Dr. MAHOMED.

Daily, at Half-past Two.

Cutaneous Diseases.—Dr. PYE-SMITH.

Tuesdays, at Twelve.

Practical Physiology.—Dr. WOOLDRIDGE.

Tuesdays, Wednesdays and Fridays, at Half-past One.

Morbid Histology.—Mr. SYMONDS.

Two days in the week.

Practical Pharmacy.

SUMMER COURSES.

The Summer Session begins May 1st and ends July 31st.

LECTURES.

Materia Medica and Therapeutics.—Dr. TAYLOR.

Mondays, Tuesdays, and Thursdays, at Two.

Midwifery and Diseases of Women.—Dr. GALABIN.

Tuesdays, Wednesdays, Thursdays, and Fridays, at Nine.

Medical Jurisprudence.—Dr. STEVENSON.

Tuesdays, Thursdays, and Saturdays, at Ten.

Clinical Medicine.—Dr. TAYLOR, Dr. GOODHART, and Dr. MAHOMED.

Wednesdays, at Half-past One.

Clinical Surgery.—Mr. LUCAS, Mr. GOLDING-BIRD, Mr. JACOBSON, and Mr. SYMONDS.

Fridays, at Half-past One.

Clinical Lectures on Diseases of Women.—Dr. HOBROCKS.

Tuesdays, at Half-past One.

Pathology.—Dr. GOODHART, *Saturdays, at Nine.*

Hygiene.—Mr. GEORGE TURNER, *Mondays and Fridays, at Half-past Eleven.*

Mental Diseases.—Dr. SAVAGE.

Tuesdays, at Eleven, and Fridays, at Half-past Ten.

Botany.—Mr. BETTANY.

Tuesdays, Thursdays, and Saturdays, at a Quarter past Eleven.

Dental Surgery.—Mr. MOON.

DEMONSTRATIONS.

Practical Chemistry.—Mr. C. E. GROVES, F.R.S.

Mondays, Wednesdays, and Fridays, Ten to One.

Operative Surgery.—Mr. LUCAS.

Mondays, Wednesdays, and Fridays, at Four.

Morbid Anatomy.

Cutaneous Diseases.

Morbid Histology.

Practical Pharmacy.

Practical Obstetrics.

Surgical Classes.

With the same arrangements as during the Winter Session.

Practical Courses and University Classes in Anatomy, Physiology, Materia Medica, Botany, Comparative Anatomy, and Natural Philosophy.

The Registrars and the Demonstrators of Anatomy and Chemistry assist Pupils in their Studies, and prepare them for their several Examinations by Special Class Instruction, during both Winter and Summer Sessions.

The Hospital contains besides the beds for Medical and Surgical Cases, wards for Obstetric and Ophthalmic cases, and other special departments.

Special Classes are held in the Hospital for Students preparing for the Examinations of the University of London and of other examining boards.

APPOINTMENTS.

The House Surgeons and House Physicians, the Obstetric Residents, Clinical Assistants, and Dressers are selected from the Students according to merit and without payment. There are also a large number of Junior Appointments, every part of the Hospital practice being systematically employed for instruction.

ENTRANCE SCHOLARSHIPS.

Open Scholarships of 125 Guineas in Classics, Mathematics, and Modern Languages. Open Scholarship of 125 Guineas in Chemistry, Physics, Botany, and Zoology.

PRIZES, &c.

The Treasurer's Gold Medal in Medicine; the Treasurer's Gold Medal in Surgery; the Gurney Hoare Prize of £25 for Clinical Study; the Beaney Prize of 30 Guineas for Pathology; the Sands Cox Scholarship of £15 per annum for three years for Physiology; the Joseph Hoare Prizes of £25 and £10; the Michael Harris Prize of £10 for Anatomy; the Mackenzie Bacon Prize of £10 for Ophthalmoscopy; the Mackenzie Bacon Prize of £15 for Nervous Diseases; the Burdett Prize for Hygiene, value £10; and four other Scholarships, varying in value from £10 to £50 each for general proficiency in Medical Study.

For Prospectus and further information apply to the Dean, Dr. F. Taylor.

ASTLEY COOPER PRIZE.

**The next Triennial Prize of Three Hundred Pounds,
Under the Will of the late SIR ASTLEY P. COOPER, Bart.,**

WILL BE AWARDED TO

THE AUTHOR OF THE BEST ESSAY OR TREATISE ON

“ Diseases and Injuries of the Nerves and their Surgical Treatment, together with the Operations performed upon Nerve-Trunks in the Treatment of various Diseases, and descriptions of the Changes which ensue in other Structures as well as in the Nerves themselves from these Operations.”

THE Condition annexed by the Testator is, “That the Essays or Treatises to be written for such Prize shall contain original experiments and observations, which shall not have been previously published, *and that each Essay or Treatise shall* (as far as the subject shall admit of) *be illustrated by preparations and by drawings*, which preparations and drawings shall be added to the Museum of Guy’s Hospital, and shall, together with the Work itself and the sole and exclusive interest therein and the copyright thereof, become henceforth the property of that Institution, and shall be relinquished and transferred as such by the successful candidate.”

And it is expressly declared in the Will “That no Physician or Surgeon, or other officer for the time being, of Guy’s Hospital or of St. Thomas’s Hospital, in the Borough of Southwark, nor any person related by blood or affinity to any such Physician or Surgeon, for the time being, or to any other Officer for the time being in either of the said Hospitals, shall at any time receive or be entitled to claim the Prize.” But, with the exception here referred to, this Prize is open for competition to the whole world.*

Candidates are informed that their Essays, either written in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy’s Hospital on or before January 1st, 1886, addressed to the Physicians and Surgeons of Guy’s Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy’s Hospital until claimed by the respective writers or their agents.

* The Prize cannot be awarded to any Essay that is the joint production of two or more authors.

GUY'S HOSPITAL REPORTS FOR 1882.

Forty-first Volume, being the Twenty-sixth Volume of the Third Series. Edited by H. G. HOWSE, M.S., and FREDERICK TAYLOR, M.D. Contains pp. 516, 2 Chromo-lithographs, 8 Plates, and 27 Woodcuts. Price to Subscribers, 6s.; to non-Subscribers, 7s. 6d. Postage free.

CONTENTS.

1. In Memoriam, Joseph Towne, Modeller to Guy's Hospital for Fifty-three Years. By Thomas Bryant.
2. A Case of Phosphorus-Poisoning which ended in Recovery under the Administration of Oil of Turpentine. By C. Hilton Fagge, M.D.
3. A Case of Symmetrical Softening of the Corpora Striata, followed by Bilateral Descending Degeneration with Secondary Anterior Poliomyelitis. By W. Hale White, M.D.
4. Exophthalmic Goitre with Mental Disorder. By George H. Savage, M.D.
5. Cases of Empyema in Children, treated by Removal of a Portion of Rib. By W. Arbuthnot Lane, B.S.
6. Abnormalities observed in the Dissecting-room of Guy's Hospital during the Sessions 1880-81 and 1881-82. By R. E. Carrington, M.D., P. Horrocks, M.D., and W. Hale White, M.D.
7. Two Cases of Pulsatile Tumour at the Root of the Neck. By C. H. Golding-Bird and F. A. Mahomed, M.D.
8. The Surgical Affections of the Tongue. By Thomas Bryant.
9. On Hemianæsthesia. By Samuel Wilks, M.D.
10. Saturnine Lunacy. By James F. Goodhart, M.D.
11. On Acute Gonorrhœal Rheumatism. By J. N. C. Davies Colley, M.C.
12. Some Remarks on the Minute Anatomy and Origin of the Enchondromata of the Salivary Glands. By W. H. A. Jacobson.
13. Report of a Case of Idiopathic Anæmia of Addison, since called Essential, Pernicious, or Progressive Anæmia, with a Commentary and Tables of Selected Cases. By P. H. Pye-Smith, M.D.
14. Poisoning by Aconitine (case of Reg. v. Lamson). By Thomas Stevenson, M.D.
15. Laboratory Notes on the Working of the Histological Class. By C. Hilton Golding-Bird.
16. Cases of Paralysis of the Abductors of the Vocal Cords. By Frederick Taylor, M.D.
17. Cases of Multiple Small Abscesses of the Liver. By R. E. Carrington, M.D.
18. Pes Valgus Acquisitus; Pes Pronatus Acquisitus; Pes Cavus. By C. Hilton Golding-Bird.
19. Lead Poisoning. By Thomas Stevenson, M.D.
20. On the Vitreous Body in its Relation to Various Diseases of the Eye. By W. A. Brailey, M.D.
21. Statistical Summary of Patients treated in Guy's Hospital during the past four years (1879—1882). By J. C. Steele, M.D.

Lists of Pupils who have obtained distinctions at the Universities, Colleges, and in the Medical School of Guy's Hospital.

J. & A. CHURCHILL, New Burlington Street.

